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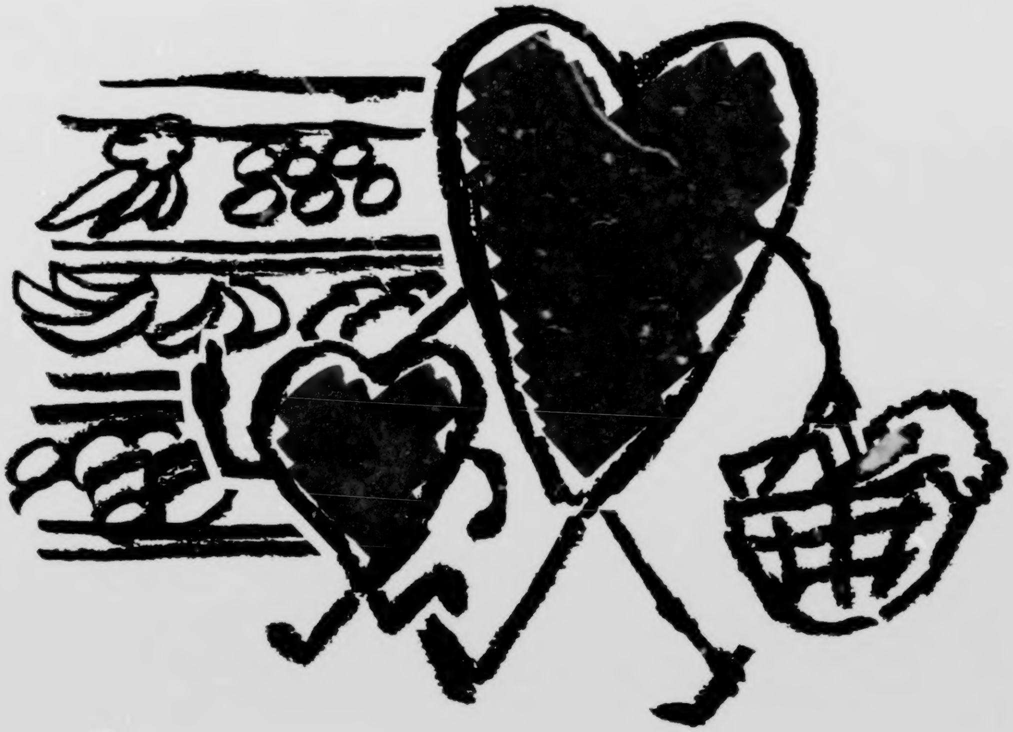
National Cholesterol Education Program

Report of the Expert Panel on

Population Strategies for Blood Cholesterol Reduction



U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES

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U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service • National Institutes of Health • National Heart, Lung, and Blood Institute
National Cholesterol Education Program

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November 1990

Dedication To

JOSEPH STOKES III, M.D.

Dr. Joseph Stokes was a member of this panel until his untimely death in June 1989. It was his characteristically clear-sighted vision of the need for population strategies to lower blood cholesterol that led to the formation of the panel, and his contributions to the formulation and execution of the panel's work were extraordinary. Dr. Stokes played a pivotal role in shaping the development of the National Cholesterol Education Program (NCEP) as a leading member of the program's Coordinating Committee. His insight, scholarship, and commitment to public health were vitally important in ensuring the early success of the NCEP. Dr. Stokes' leadership and effective advocacy on behalf of the NCEP mirrored his dedication and monumental contributions to the prevention of cardiovascular disease in the United States. We dedicate this report to the memory of Dr. Joseph Stokes with respect and affection.

National Cholesterol Education Program

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I. Overview and Summary

Coronary heart disease (CHD) is a major public health problem in the United States. Despite substantial success in reducing premature deaths from CHD in the past two decades, this disease continues to kill more than 500,000 Americans annually. About 1,250,000 Americans suffer myocardial infarctions each year, and millions more have angina pectoris. In addition, significant degrees of asymptomatic CHD are very common in our population. In addition to its impact on the Nation's health, CHD costs the U.S. economy over $50 billion annually.

Coronary heart disease is the result of atherosclerosis, in which deposits of cholesterol and other lipids, along with cellular reactions, thicken artery walls. This process gradually reduces the caliber of the artery and restricts blood flow. Inadequate blood flow may cause injury to or death of tissue beyond the site of reduced flow; in the coronary arteries, this leads to myocardial infarction or sudden death.

Many factors influence not only whether a person will develop CHD but also how rapidly atherosclerosis progresses. Genetic predisposition, gender, and advancing age are recognized factors over which we have little control. High blood cholesterol, cigarette smoking, high blood pressure, excessive body weight, and long-term physical inactivity are also key risk factors over which we have considerable control. Control of each of these factors is important in the prevention of CHD. People with diabetes may also be able to avoid or delay vascular disease by controlling the other risk factors.

High blood cholesterol levels clearly play a causal role in CHD. This conclusion is based on experimental laboratory work, extensive clinical and pathological research, and numerous epidemiologic studies over the past several decades. The magnitude of the problem posed by elevated blood cholesterol levels is very clear. High CHD rates occur among people with high blood cholesterol levels of 240 mg/dL (6.21 mmol/L) or above. However, an even larger number of *cases* occurs in Americans with blood cholesterol levels below 240 mg/dL. It is important to note that the average cholesterol level for the adult U.S. population is about 210 mg/dL. Furthermore, approximately 55 percent of adult Americans have cholesterol levels at or above 200 mg/dL.

For these reasons, two kinds of strategies are needed: patient-based strategies, which seek to help those with the highest blood cholesterol levels, and population-based strategies, which seek to reach all Americans. The population approach aims both to lower the blood cholesterol level of individuals and to reduce the average cholesterol level throughout the population. When both approaches are used, the effects are synergistic.

Two important types of research provide ground for optimism that CHD morbidity can be reduced, and that the trend of reduced CHD death rates will continue or accelerate. First, studies consistently indicate that reducing blood cholesterol can reduce the likelihood of developing or dying from CHD. Dietary factors in individuals and in entire populations have important effects on blood cholesterol levels (although genetic makeup, expressed, in part, as a low level of HDL-cholesterol or as a high level of LDL-cholesterol, also plays a major role). Second, this report clearly indicates that many effective strategies exist for helping Americans develop more healthful eating patterns and lifestyles.

The Population Panel of the National Cholesterol Education Program offers a set of recommendations designed to help healthy Americans lower their blood cholesterol levels through changes in eating patterns and thus reduce their likelihood of developing CHD. Recognizing that Americans' food consumption is influenced by many factors, the panel directs its recommendations to individuals, to special population groups, to health professionals, to the many components of the food industry, to relevant government agencies, and to public and private education systems. In addition, the panel's recommendations address cholesterol screening, and the need for continued research and evaluation as eating patterns change, blood cholesterol levels decrease, and CHD continues to decline.

The panel recommends the following nutrient intakes for healthy Americans:

- Less than 10 percent of total calories from saturated fatty acids.
- An average of 30 percent of total calories or less from all fat.
- Dietary energy levels needed to reach or maintain a desirable body weight.
- Less than 300 mg of cholesterol per day.

Each is intended to be achieved by an individual as an *average* of nutrient intake over several days.

These recommendations concerning nutrient intakes are appropriate for the general population, including healthy *women* and *individuals 65 years of age and older*.

As *healthy children* join in the eating patterns of others in the family, usually at about 2 years of age or older, they should follow the recommended nutrient intake and eating patterns.

To achieve more healthful eating patterns, the panel recommends that healthy Americans select, prepare, and consume foods that contain lower amounts of saturated fatty acids, total fat, and cholesterol; choose a variety of foods to ensure recommended intakes of carbohydrates, protein, and other nutrients; and consume only enough calories to maintain desirable weight.

The panel also makes recommendations for other groups:

Health professionals should both practice and advocate the recommended eating patterns; ensure that education of future health professionals includes appropriate nutrition education; and work with industry, government, voluntary groups, and health care agencies to facilitate adoption of the recommended eating patterns.

The food industry, food and animal scientists, and food technologists should increase efforts to design, modify, prepare, promote, label, and distribute good-tasting, safe foods that are lower in saturated fatty acids, total fat, and cholesterol.

Government agencies should provide consistent, coordinated nutrition statements and policies emphasizing low saturated fatty acid, low-fat, and low-cholesterol eating patterns; should expand and standardize food labeling requirements to identify clearly the content of saturated fatty acids, total fat, cholesterol, and total calories; and should take other steps to improve the consumer comprehension necessary to achieve the recommended eating patterns.

Educational programs at all levels should incorporate curricula that emphasize the background, benefits, and methods of achieving eating patterns that are lower in saturated fatty acids, total fat, and cholesterol. This recommendation includes elementary through high schools, vocational programs (especially in culinary arts), colleges, universities, and health professional schools.

Measurement of blood cholesterol, followed by appropriate education and counseling, is best initiated in the health care setting; but in specific circumstances and especially for selected segments of hard-to-reach population groups, public screening for blood cholesterol, when carried out with high quality standards, is appropriate.

Research and surveillance must be ongoing to develop new information concerning diet, blood lipids, and CHD; the development of better data bases concerning food composition, food consumption patterns, illness rates, food product development, and nutrition education and communication is critical.

Implementation of these recommendations will promote adoption of eating patterns that will help most Americans lower their levels of blood cholesterol. The result, an approximate reduction of 10 percent or more in the average blood cholesterol level of the U.S. population, will lead to an approximate reduction of 20 percent or more in coronary heart disease and, in consequence, to significant improvement in the health and quality of life of Americans.

II. Background and Introduction

Cholesterol was identified many years ago as a prominent chemical component of the atherosclerotic lesion in human arteries. Following those early observations, a large base of scientific evidence has accumulated linking dietary saturated fatty acid (SFA) and cholesterol intake, blood cholesterol, atherosclerosis, and coronary heart disease (CHD). Animal, biochemical, genetic, metabolic, clinical, and epidemiologic research have all made important contributions to understanding these relationships. Clinical intervention trials have shown reduced CHD rates when cholesterol levels are reduced. While it is apparent that individuals with high blood cholesterol levels are at high risk of CHD, a larger number of individuals with more moderately elevated cholesterol also have increased risk of coronary events; such individuals comprise a substantial proportion of the adult U.S. population. From these observations, it follows that a two-pronged strategy is appropriate to address the high prevalence of CHD in Americans. The patient-based approach seeks to identify those at highest risk and to treat them to lower their blood cholesterol level. The population approach seeks to influence the population at large to alter current eating patterns, and thereby to lower individual and average population levels of blood cholesterol. These two approaches are complementary means of achieving lower rates of CHD and better health of the public. This report deals with the population approach.

In 1984, the National Institutes of Health (NIH) convened a Consensus Development Conference on Lowering Blood Cholesterol to Prevent Heart Disease. Among the recommendations from this conference were identification and treatment of individuals with high-risk cholesterol levels, changes in eating patterns for members of the general public, and the creation and implementation of a national cholesterol education program, which the National Heart, Lung, and Blood Institute (NHLBI) had begun to plan in early 1984. The National Cholesterol Education Program (NCEP) was launched in November 1985. The goal of the NCEP is to reduce the prevalence of elevated blood cholesterol in the United States, thereby contributing to the reduction of CHD morbidity and mortality. The NCEP is designed to reach and influence both health professionals and the public. This program provides an opportunity for individuals to assume an active role in reducing their risk of CHD. Under the direction of a multidisciplinary coordinating committee representing a variety of agencies and organizations, the program has accelerated the process of increasing public and professional awareness of the importance of reducing elevated blood cholesterol levels. The NCEP has issued reports developed by its Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel or ATP) and its Laboratory Standardization Panel (LSP). The present report has been developed by a third panel, the Population Panel, which has conducted an intensive review of the scientific basis for making blood cholesterol-lowering recommendations, especially eating pattern recommendations, to the general public. A fourth panel will report later on blood cholesterol reduction in children and adolescents.

A. Coronary Heart Disease

Atherosclerosis is a complex disorder, the development of which involves the interplay of many factors. Blood cells such as monocytes and platelets, the blood coagulation system, endothelial cell damage, and deranged function of arterial wall macrophages and smooth muscle cells all contribute to the atherosclerotic process. Many other risk factors play important roles. The process involves the deposition of cholesterol in arterial walls and progresses with cellular proliferation, fibrous tissue deposition, and further cholesterol deposition, creating progressively larger atherosclerotic plaques. As atherosclerosis progresses, the opening in the artery through which blood flows is gradually reduced in size. The degree of arterial narrowing may be abruptly increased by thrombosis, by plaque disruption, or by smooth muscle spasm. Closure of an artery may cause injury and death of tissue beyond the site of blockage. When a coronary artery closes, myocardial infarction or sudden death is a common outcome.

Approximately 1,250,000 Americans each year suffer a myocardial infarction. More than 500,000 die annually from CHD. More than 6,000,000 Americans have symptomatic CHD, while it is estimated that nearly 70 percent of adult Americans have some degree of atherosclerotic narrowing of their coronary arteries. From a public health perspective, CHD is a major health problem. The economic costs have been estimated to be over $50 billion annually for illness care and lost earnings and productivity related to CHD.

Although the precise causes of atherosclerosis have not been identified fully, clinical, epidemiological, animal, and biochemical evidence demonstrate that many major factors contribute to the process. Three of these factors—gender, advancing age, and genetic composition—cannot be changed, but their effects can be mitigated.

Several other important risk factors for CHD are modifiable and provide opportunities for individuals and populations to reduce their likelihood of developing CHD. The most important are high blood cholesterol, high blood pressure, and cigarette smoking. Numerous epidemiologic studies show that the smoking habit increases the chances of developing CHD, as well as the probability of lung cancer or emphysema. The risk of both CHD and lung disease decreases with smoking cessation. High blood pressure accelerates the atherosclerotic process; control of high blood pressure reduces the likelihood of stroke and probably decreases the incidence of CHD.

Other risk factors have also been identified. Three are of particular note: overweight, diabetes mellitus, and a sedentary lifestyle. Maintaining or achieving a desirable weight is important both by itself (because excess weight is probably an independent risk factor for CHD) and because people who have a desirable body weight are less likely to have high blood cholesterol, a low high density lipoprotein (HDL) cholesterol level, or high blood pressure and are also less likely to develop diabetes mellitus than obese people. It may be possible to prevent many cases of non-insulin-dependent diabetes mellitus by avoiding obesity; furthermore, there is hope that control of the three major modifiable CHD risk factors will help diabetics avoid many vascular complications. Moderate, regular aerobic exercise throughout life is also important, in part because it enhances cardiovascular fitness and facilitates control of high blood pressure and excess body weight, and in part because of its generally favorable effect on HDL-cholesterol.

As has been reviewed extensively in section V of this report, entitled Scientific Evidence for Recommendations Affecting the General Public, consistent evidence from multiple avenues of research implicates high blood cholesterol in the causation of atherosclerosis and CHD. Both genetic and dietary factors influence blood cholesterol levels. Saturated fatty acids (SFA), cholesterol, and excessive calories in the diet tend to elevate the blood cholesterol level. There is now conclusive evidence that lowering elevated blood cholesterol decreases CHD risk; this has special importance for dietary recommendations.

The NCEP represents a major effort to reduce CHD rates in the United States by helping to lower elevated blood cholesterol levels, in part through favorable alterations in eating patterns. Together, the National High Blood Pressure Education Program, federally sponsored smoking education programs, and the NCEP are designed to reduce the three major modifiable risk factors for CHD and improve the health of the American public.

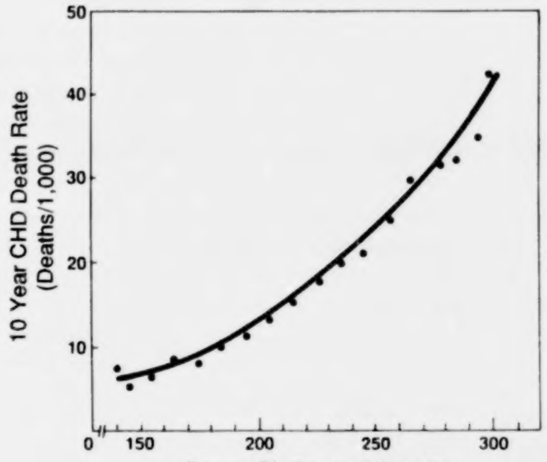
B. Rationale for Both a Population Approach and a High-Risk Approach to Blood Cholesterol

Epidemiological, clinical, and experimental evidence clearly demonstrate that the likelihood that a person will develop or die from CHD is directly related to the level of blood cholesterol. This relationship is well illustrated by the 10-year followup experience of men, initially ages 35 to 57 years, screened for the Multiple Risk Factor Intervention Trial (MRFIT).[1] As shown in figure 1, using data from MRFIT, the relationship is continuous and the risk increases with rising cholesterol levels. The risk of dying from CHD increases slowly between 150 mg/dL (3.88 mmol/L) and 200 mg/dL (5.17 mmol/L) and more rapidly when the cholesterol level exceeds about 200 mg/dL (5.17 mmol/L). For those individuals with cholesterol levels between 240 mg/dL (6.21 mmol/L) and 300 mg/dL (7.76 mmol/L), the likelihood of CHD is increased up to fourfold above that for those with levels below 200 mg/dL (5.17 mmol/L). Similar relationships between blood cholesterol and CHD have been found in many other studies.

In the United States, as in many other nations of the world, a majority of people have a blood cholesterol level high enough to increase significantly the likelihood of CHD. Thus, data from the National Health and Nutrition Examination Survey, known as NHANES II (1976-80) (figure 2, page 10), showed the mean cholesterol level for the adult U.S. population to be about 210-215 mg/dL (5.43-5.56 mmol/L).[2] Approximately 30 percent of adult Americans had cholesterol levels of 200-239 mg/dL (5.17-6.18 mmol/L), and more than 25 percent had levels of 240 mg/dL (6.21 mmol/L) and above. Figure 1 shows the high CHD death rate of those in this upper 25 percent of the cholesterol distribution. Despite the lower relative risk of the 75 percent of the population with blood cholesterol levels below 240 mg/dL (6.21 mmol/L), this group experiences about 60 percent of all CHD deaths, reflecting both the larger number of people at risk and the effects of mildly to moderately elevated cholesterol levels as well as the influence of other risk factors. The fact that risk of CHD is by no means restricted to those with high blood cholesterol levels supports the need for a combined population and high-risk approach to CHD prevention.

Based on the scientific evidence of increased CHD risk with elevated blood cholesterol, along with the evidence that risk can be reduced by lowering high blood cholesterol levels, the ATP of the NCEP issued a series of recommendations for a strategy of detecting and treating individuals with a high blood cholesterol level.[3] This panel, building on the work of the Consensus Development Conference on Lowering Blood Cholesterol to Prevent Heart Disease, classified blood cholesterol levels for adults. Levels under 200 mg/dL (5.17 mmol/L) are termed "desirable." Levels between 200 mg/dL and 239 mg/dL (5.17 and 6.18 mmol/L) have been classified "borderline-high," while levels of 240 mg/dL (6.21 mmol/L) and

Figure 1
Relationship Between Serum Cholesterol Level and CHD Death Rate (MRFIT Screenees)



From 361,662 Men Screened for MRFIT Program

above have been designated as "high." The ATP has presented a comprehensive set of recommendations for intervention in individuals with cholesterol levels above the desirable range. These recommendations emphasize that after initial screening for total cholesterol level, therapeutic decisions should be based on the level of low density lipoprotein (LDL) cholesterol (see table 1). Adult Americans with LDL-cholesterol levels that are persistently high-risk (or borderline-high-risk if definite CHD or two or more of the other major CHD risk factors listed in table 2 are present) should follow the recommendations of the ATP of the NCEP.

The ATP set forth detailed recommendations indicating that dietary therapy is the mainstay of treatment. Drug therapy should generally be considered only after 6 months of intensive dietary therapy reinforced by the help of a health professional with special nutrition expertise (e.g., a registered dietitian), and then only if the LDL-cholesterol level remains significantly elevated. This conservative approach to drug therapy is wise because drugs can not only bring benefits but can also have deleterious effects.

The potential impact of the recommendations of the ATP on the blood (serum) cholesterol distribution of the adult U.S. population is indicated conceptually by the broken line in figure 3, page 10. These recommendations of the ATP should, over the years to come, reduce the proportion of individuals in the United States with high cholesterol levels (240 mg/dL [6.21 mmol/L] and above), and should also reduce the proportion of individuals with borderline-high cholesterol levels (200-239 mg/dL [5.17-6.18 mmol/L]). Even in the event that the high-risk approach recommended by the ATP is largely effective, however, many people will remain at risk of CHD.

The recommendations from the Population Panel of the NCEP follow. They are intended to extend the benefits of cholesterol lowering to the population as a whole by promoting adoption of eating patterns that will help lower the blood cholesterol levels of most Americans. In turn, these eating patterns should help to shift the curve representing the population distribution of cholesterol to the left, as shown in figure 4, page 11. As the recommended eating patterns become widely adopted, the average blood cholesterol level should decrease by approximately 10 percent or greater.

Table 1

Recommendations of the Adult Treatment Panel of the National Cholesterol Education Program for Classification of Patients

Classification Based on Total Cholesterol	Classification Based on LDL-Cholesterol
<200 mg/dL (<5.17 mmol/L) Desirable Blood Cholesterol	<130 mg/dL (<3.36 mmol/L) Desirable LDL-Cholesterol
200-239 mg/dL (5.17-6.18 mmol/L) Borderline-High Blood Cholesterol	130-159 mg/dL (3.36-4.11 mmol/L) Borderline-High-Risk LDL-Cholesterol
≥240 mg/dL (≥6.21 mmol/L) High Blood Cholesterol	≥160 mg/dL (≥4.13 mmol/L) High-Risk LDL-Cholesterol

Source: NCEP ATP, January 1988.[3]

Table 2

CHD Risk Factors Other Than LDL-Cholesterol

- **Male sex**
- **Family history of premature CHD** (definite myocardial infarction or sudden death before age 55 in a parent or sibling)
- **Cigarette smoking**
- **Hypertension**
- **Low HDL-cholesterol concentration** (below 35 mg/dL confirmed by repeat measurement)
- **Diabetes mellitus**
- **History of definite cerebrovascular or occlusive peripheral vascular disease**
- **Severe obesity** (≥30% overweight)

Source: NCEP ATP, January 1988.[3]

The anticipated combined and compatible effects of the recommendations from the Adult Treatment Panel and from the Population Panel of the NCEP are estimated in figure 5, page 11. Most Americans would have a desirable blood cholesterol level. Far fewer Americans would require drug therapy for persistently high blood cholesterol. The anticipated effect would be to reduce the morbidity from CHD and to produce a continued or accelerated decline in the mortality rate from CHD in the United States.

C. Guidelines From Other Authoritative Sources

1. International

The work of many scientists has shown that atherosclerosis and CHD constitute a major health problem afflicting numerous nations of the world. The linkage of CHD to national eating patterns has been established. There is widespread international consensus, based upon repeated reviews of the scientific evidence, that the average population intakes of total fat, SFA, dietary cholesterol, and food energy are excessive in many countries and should be reduced. As a result, since 1973 more than 40 groups of scientists and health policy makers from other nations have recommended changes in population eating patterns with the purpose of reducing blood cholesterol. Most have focused on the prevention of CHD. These recommendations have been summarized by Truswell (1983),[4] and in the report of the National Research Council (NRC), *Diet and Health: Implications for Reducing Chronic Disease Risk.*[5] A World Health Organization (WHO) study group in 1989 proposed "population nutrient goals" to control noncommunicable diseases and suggested that these be introduced as part of nutrition policy in all countries[6,7]. These nutrient goals are remarkably similiar to those of the NRC 1989 and NCEP 1990.

Cholesterol Distribution in the U.S. Population (Males/Females Combined, Ages 20-74, NHANES II 1976-80),[2] and Potential Changes in the Distribution

Figure 2

Population distribution of serum cholesterol values. The borderline-high and high cutoff levels are shown as dotted lines to indicate the proportions of the population above or below 200 or 240 mg/dL (5.17 or 6.21 mmol/L).

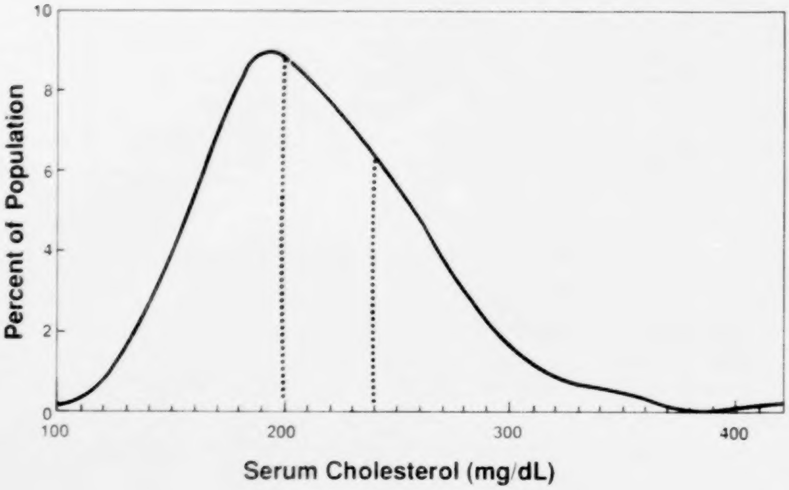
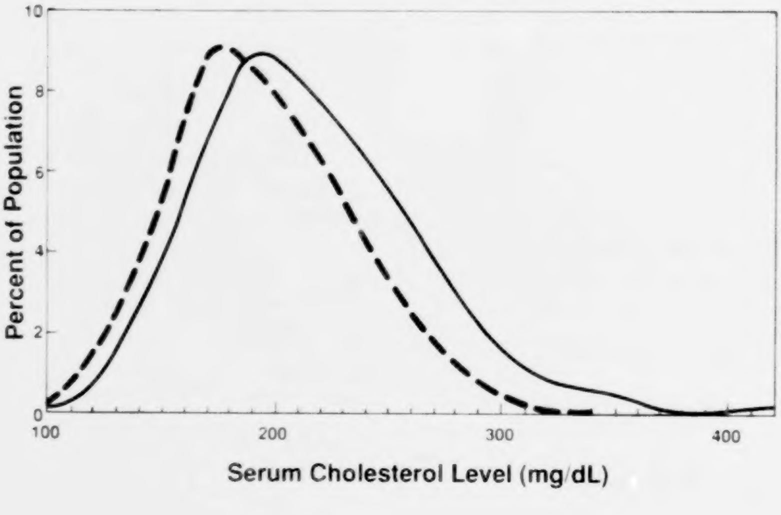


Figure 3

Expected shift in population distribution of serum cholesterol values with widespread application of ATP guidelines. The dotted-dashed line represents an estimate of the effect of treating many people with elevated cholesterol levels.



Cholesterol Distribution in the U.S. Population (Males/Females Combined, Ages 20-74, NHANES II 1976-80),[2] and Potential Changes in the Distribution

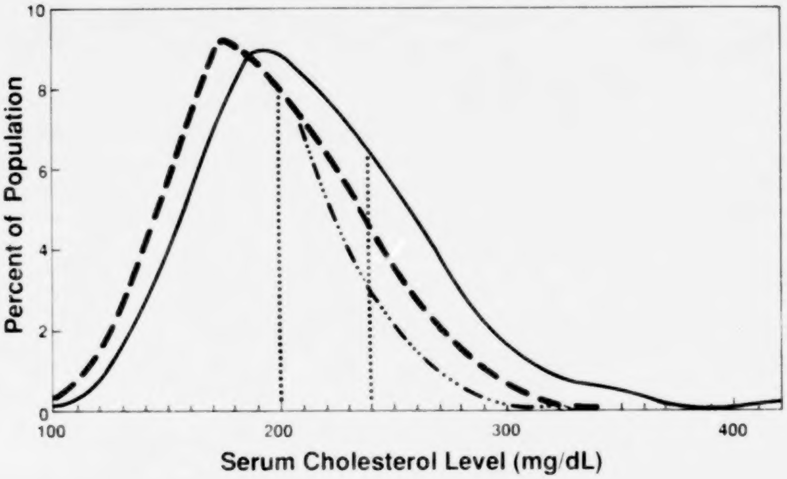


Figure 4

Expected shift in population distribution of serum cholesterol values if the recommendations of the Population Panel result in a 10% decrease in blood cholesterol of Americans. The dashed line shows the effect of the recommendations.

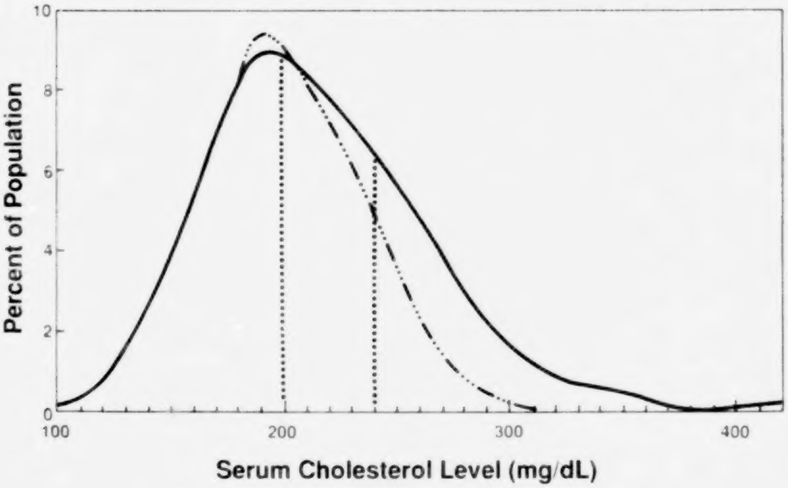


Figure 5

Anticipated combined effects of the recommendations of the ATP (dotted-dashed line) and the Population Panel (dashed line).

2. United States

Over the years, many reports linking CHD and diet have appeared in the United States. These have included several diet statements from the American Heart Association (AHA) issued between 1961 and 1988, the 1970 and 1984 reports of the Inter-Society Commission for Heart Disease Resources concerning primary prevention of atherosclerotic diseases,[8,9] the Bethesda Conference on Prevention of Coronary Heart Disease,[10] and the recent NRC report on diet and health.[5]

The dietary recommendations issued since 1983 by major health organizations in the United States are summarized in table 3. There are common themes—recommendations for a lower intake of SFA and total fat, a reduction in dietary cholesterol, an increased intake of complex carbohydrates, and the control of obesity. The advice given is similar from all these organizations, although some groups are more specific than others. Several recommend reductions of total fat intake to 30 percent or less of calories, of SFA to less than 10 percent of calories, and of dietary cholesterol to less than 300 mg per day.

Table 3

Dietary Recommendations Related to Blood Cholesterol Levels by United States Organizations Since 1983

Agency/Report	**Year**	**Audience**	**Saturated Fat**	**Total Fat**	**Cholesterol**	**Carbohydrate**	**Body Weight**
USDA/DHHS Dietary Guidelines for Americans	1985	Public	Avoid too much	Avoid too much	Avoid too much	Avoid too much sugar; Increase starch & fiber	Maintain desirable weight
NCEP-ATP Report of the Expert Panel on. . .High Blood Cholesterol in Adults	1987						
Step 1		Adult patients with high blood cholesterol	<10%	≤30%	<300mg/day	50-60%	Achieve & maintain desirable weight
Step 2			<7%	≤30%	<200mg/day	50-60%	
AHA Dietary Treatment of Hypercholesterolemia Steps 1 & 2 for patients identical to NCEP Steps 1 & 2	1988						
AHA Dietary Guidelines for Healthy American Adults	1988	Public	<10%	<30%	≤300 mg/day	50-55% with emphasis on complex carbs.	Maintain best body weight
AHA Diet in the Healthy Child	1983	Children	≤10%	Approx. 30%	≤300 mg/day	Approx. 55%	Caloric intake based on growth rate & activity level and to maintain desirable body weight
AAP Prudent Life-style for Children: Dietary Fat and Cholesterol	1986	Children	Decrease with moderation	30-40%	Decrease with moderation		Maintain ideal body weight
DHHS The Surgeon General's Report on Nutrition and Health*	1988	Public	Reduce	Reduce for most	Reduce	Increase complex carbs.; Limit sugars	Achieve & maintain desirable weight
NRC Designing Foods: Animal Product Options in the Marketplace**	1988	Public	≤10%	≤30%	≤300 mg/day		Achieve & maintain desirable weight
NRC Diet and Health: Implications for Reducing Chronic Disease Risk	1989	Public	<10%	≤30%	<300 mg/day	>55% with ≥6 servings grains & legumes/day, ≥5 servings fruits & vegetables per day	Balance food intake with physical activity to maintain body weight

* A review supporting USDA/DHHS (1985) recommendations.

** The report accepted these target levels from other authorities; it also made recommendations on marketing and policy issues and research imperatives.

III. Conclusions of the Population Panel

After carefully considering the extensive scientific evidence linking blood cholesterol, atherosclerosis, CHD, and diet, this panel concludes that excessive intakes of saturated fatty acids, total fat, and dietary cholesterol, together with excessive body weight, all contribute importantly to biologically unnecessary and undesirable elevations of blood cholesterol. The panel also reaffirms the conclusion of previous panels (AHA, ATP, NRC) that elevated levels of blood cholesterol produce a high prevalence of severe atherosclerosis in coronary arteries, resulting in a high incidence of CHD and premature death. Accordingly, the panel concludes that it is important for Americans to change their eating patterns to reduce the average intakes of saturated fatty acids, total fat, and dietary cholesterol, and to eliminate excess body weight.

The panel concludes that changing eating patterns will influence blood cholesterol levels and that eating patterns can be changed, while preserving the nutritional adequacy, variety, and affordability of good-tasting food. The panel regards reduction of dietary saturated fatty acid intake from both animal and vegetable sources as being of greatest importance. The work of the National Research Council[11] concerning the potential for reduction of fat in foods of animal origin indicates the feasibility of change in saturated fatty acid content of foods. Furthermore, the panel agrees that observance of the Dietary Guidelines for Americans issued by the U.S. Department of Agriculture (USDA) and the Department of Health and Human Services (DHHS) can help promote health for individuals beyond the age of 2 years.[12] Although there are challenges to implementing some of the recommended changes, the panel believes that the potential benefits outweigh the expenditure of resources needed to achieve these changes. The panel believes that eating patterns of most Americans can change through a process of combining education for the public and actions by the many sectors of society that influence the availability, purchase, preparation, and consumption of foods.

The panel agrees with estimates that the health benefits to be expected for the average American, based upon available evidence, approximate a 2 percent reduction in CHD risk for each 1 percent reduction in blood cholesterol level.

It is from these perspectives that the Population Panel of the NCEP offers recommendations to help individuals in the United States lower their blood cholesterol levels.

IV. Recommendations

These recommendations are designed to encourage and assist each individual to adopt eating patterns to lower his or her blood cholesterol level and to reduce the number of people in the United States who develop or die from CHD. A blood cholesterol level under 200 mg/dL (5.17 mmol/L) is termed desirable for adults. To help Americans learn whether they have an elevated blood cholesterol level, this panel concurs with the ATP recommendation that all adult Americans should have their blood cholesterol level measured at least every 5 years. The average level of blood cholesterol in the U.S. adult population is approximately 210-215 mg/dL (5.43-5.56 mmol/L). It is anticipated that the recommended changes in eating patterns will result in a reduction of 10 percent or greater in the average person's blood cholesterol level and shift the population distribution curve for blood (serum) cholesterol values as shown in figure 4, page 11.

The nature of the food consumed by Americans is influenced by many factors. These recommendations are intended to provide guidance to many segments of the food production-consumption chain. Success will require adoption of these recommendations by all healthy Americans, health professionals, the food industry, educators at all levels, mass media, and many government agencies.

Accordingly, the panel makes the following recommendations for the *entire population of healthy Americans*. This population approach aims to promote healthful food choices and to make good-tasting food, lower in saturated fatty acids and cholesterol, more widely available to all Americans. Those whose blood cholesterol levels are higher than desirable can be expected to pay the most attention and to benefit the most from these recommendations. In addition, most individuals with desirable cholesterol levels can participate in the recommended eating patterns, and expect to lower CHD risk. The recommendations in this report are compatible with the Dietary Guidelines for Americans as set forth by the USDA and the DHHS.

A. Nutrient Intake

The panel concludes that health and nutrition professionals, the food industry, and the general public need specific guidance concerning intake of certain nutrients.

RECOMMENDATION A. 1: The panel recommends the following pattern of nutrient intake for all healthy Americans*:

- **Less than 10 percent† of total calories from saturated fatty acids.**
- **An average of 30 percent of total calories or less from all fat.†**
- **Dietary energy (calorie) levels needed to reach or maintain a desirable body weight.**
- **Less than 300 mg of cholesterol per day.**

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[*] Concerning the very young, as healthy children join in the eating patterns of others in the family, usually at about 2 years of age or older, they should follow the recommended nutrient and eating patterns.

[†] Each gram of fat contains 9 Kilocalories (often called calories). A person consuming 1,500 calories daily should consume no more than 450 calories (1,500 calories x 0.30 = 450 calories) derived from all fat. At 9 calories per gram, less than 50 grams of fat (450 calories ÷ 9 calories/gram = 50 grams) should be consumed on the average day, and less than one-third or 17 grams of the 50 gram total should be from saturated fatty acids (1,500 calories x 0.10 = 150 calories; 150 calories ÷ 9 calories/gram = <17 grams).

Because food intake varies from day to day, these recommendations are meant to be achieved by each individual as an average of nutrient intake over several days.

The nutrient intakes recommended are critical to health-promoting eating patterns. They are essentially the same as those recommended by the American Heart Association, the National Research Council, and in the Step-One therapeutic diet of the ATP of the NCEP. The eating patterns that will be required in order to achieve these nutrient intakes by the public are to be attained gradually over time. Ultimately, changes in the food system will facilitate these changes in population-wide eating patterns. By contrast, the Step-One diet is intended to treat individuals who are at high risk of CHD because of substantially elevated blood cholesterol levels, through intensive nutritional counseling in a clinical setting, and with the intent of achieving a rapid and certain response. What makes the Step-One diet therapeutic for those at high risk is prescription in a medical setting along with the monitoring and followup offered by health professionals. The same eating pattern is suitable for adoption by the population at large in order to achieve a population-wide reduction of blood cholesterol. It is the intent of the recommendations of the Population Panel to foster healthful eating for Americans, young and old.

1. Rationale for Nutrient Intake Recommendations

This recommended pattern of nutrient intake is intended for healthy individuals. Strong scientific evidence indicates that attainment of each of the four recommendations will help most individuals lower their blood cholesterol and, in turn, reduce their probability of developing CHD.

The specific nutrient intake recommendations are based on the scientific evidence, but the exact numerical values reflect both science and pragmatism. Thus, there is virtually unanimous agreement that SFA intake should be reduced. Most agree that the science base makes less than 10 percent of calories from SFA an appropriate level, although there is no precise guidance as to which level under 10 percent is best. For the sake of practicality, consistency, and comprehensibility, the phrase "less than 10 percent" has been chosen. A similar process led to recommendations for total fat and cholesterol intakes. The available knowledge concerning the mechanisms through which these nutrients affect blood cholesterol is summarized in section V, Scientific Evidence for Recommendations Affecting the General Public.

The panel's rationale for each of the recommended nutrient intakes follows.

a. Fat Intake

Most of the U.S. population should reduce total dietary fat intake—by an amount that varies according to how much fat is currently consumed—to help achieve and maintain desirable body weight and to reduce the risk of certain diseases, particularly CHD. (See table 3, page 13, for a summary of recommendations from other authoritative groups.) Different kinds of fatty acids have different effects on blood cholesterol levels. In general, SFA raise blood cholesterol levels. Polyunsaturated fatty acids (PUFA) of the omega-6 series lower blood cholesterol levels, as do monounsaturated fatty acids (MUFA) and omega-3 PUFA, when substituted for SFA.

Total Fat. The panel recommends a target for individuals of an average of 30 percent of total calories or less from total fat. The percentage of calories from total fat intake, independent of the relative content of the different types of fatty acids, does not determine the level of blood cholesterol or CHD risk. As stated in the NRC report on *Diet and Health: Implications for Reducing Chronic Disease Risk*, "Fat intake should be reduced by curtailing the major sources of dietary fats rather than by eliminating whole categories of foods."[5] It is not necessary to resort to very low fat diets (e.g., 10-20 percent of total calories) to achieve a maximal reduction of blood cholesterol levels through dietary means, provided that intakes of saturated fatty acids are kept low. Therefore, the panel accepts a target for individuals of an average of 30 percent or less of calories from fat. The panel does not recommend severe reduction of total fat intake; however, a limit to total fat intake is a rational part of a program aimed at reducing the risk for CHD because such a limit facilitates reduction of saturated fatty acid intake, and facilitates maintenance of desirable body weight. There is also evidence that suggests that a diet lower in fat may prevent certain types of cancer.[5,13]

Survey data indicate that for children ages 1 to 5 years, the average proportion of calories derived from fat was 34 percent; for women ages 19 to 50 years, 37 percent; and for men ages 19 to 50, 36 percent.[14-17] Thus, to achieve an average intake of 30 percent of calories or less from fat, reductions in calories from fat of at least 4-7 percent of total calories will be required, on average, for most Americans. For many individuals with fat intakes that are higher than average, however, an even greater reduction in fat intake will be necessary.

Saturated Fatty Acids. The major decrease in total fat should be in calories from SFA. Data from the USDA[14-17] indicate that SFA, as a proportion of calories, average about 13.2 percent in the diets of adults ages 19 to 50 years and 13.9 percent in those of children ages 1 to 5 years. Thus, the average reduction in the percentage of calories from SFA that is required to achieve the recommended nutrient intake is at least 3-4 percent. Individuals currently deriving higher percentages of calories from SFA will need to make greater reductions.

The greater the decrease in SFA intake, the greater will be the decline in blood cholesterol levels. Therefore, the reduction in saturated fatty acids in the diet should be as great as possible, consistent with palatability and practicality. This panel recommends a target of less than 10 percent of calories from SFA as both practical and palatable for Americans.

Other fatty acids. The remaining fatty acids, up to 20 percent of total calories, should come from unsaturated fatty acids. PUFA can provide up to, but no more than, 10 percent of total calories. The average intake of omega-6 PUFA in the American diet is about 7 percent of total calories,[5,14-17] which is an acceptable intake. Fish oil supplements generally do not reduce blood cholesterol levels. Omega-3 PUFA, however, reduce blood cholesterol when substituted for SFA. The other major type of unsaturated fatty acids, MUFA, should provide the remaining dietary fat. The current average intake of MUFA for the population is approximately 14-16 percent of calories. A slight reduction in average MUFA intake can be expected because foods of animal origin containing SFA often also have substantial amounts of MUFA.

Other Nutrients. Reducing fat intake will reduce energy intake unless other nutrients replace fat. Two other major macronutrients, protein and carbohydrates, as well as many micronutrients, are of importance. The panel recommends that the intake of carbohydrates be increased to 50-60 percent of total calories, primarily by increasing the intake of complex carbohydrates. This can be achieved by increasing the intake of vegetables and fruits, and of breads, legumes, and whole grain cereals. These plant products generally contain low levels of fat and are good sources of several vitamins, minerals, and dietary fiber. Protein intake should not be increased to compensate for the decrease in calories derived from fat. The recommended intake of protein is between 10 percent and 20 percent of calories. In general, average protein intake by adults in the United States considerably exceeds the recommended dietary allowance (RDA), which is 0.8 g/kg of desirable body weight for adults. Overall, the diet consumed should meet the RDA's for all other nutrients.

b. Body Weight

Body weight is influenced by many factors; energy balance between caloric intake and caloric expenditure largely dictates whether excess body fat accumulates. Many health problems are related to obesity. Obese people are more likely to have elevated blood cholesterol and triglyceride levels, a reduced level of HDL-cholesterol, high blood pressure, and adult-onset diabetes mellitus, and are more likely to develop CHD independently of these other factors. Loss of excess weight and maintenance of a desirable body weight is an important way of reducing blood cholesterol levels. The exact dietary energy (calorie) level needed to attain or maintain desirable body weight must be individually determined from observed changes in body weight.

c. Cholesterol Intake

Many studies have shown that dietary cholesterol raises the blood cholesterol level. It has been estimated that with a 2,500 calorie diet, for every 100 mg/day decrease in cholesterol intake, the blood cholesterol will decrease by about 4 mg/dL.[18] This response holds even at low intakes, and thus, the lower the cholesterol intake, the lower will be the blood cholesterol on the average. There appears to be considerable inter-individual variability in response of blood cholesterol to dietary cholesterol intake. Based on the observed effects of dietary cholesterol intake on blood cholesterol levels, the panel concludes, as have many investigators and expert panels, that high dietary cholesterol intake contributes to the development of atherosclerosis and increased CHD risk in the American population and should be reduced. Data from the USDA[14-17] indicated that the average daily intake of dietary cholesterol is 304 mg and 435 mg for women and men, respectively. The Population Panel recommends an intake of less than 300 mg of cholesterol per day.

B. Eating Patterns

The nutrient intakes recommended in this report are consistent with the Dietary Guidelines for Americans as set forth by the USDA and the DHHS.[12] These guidelines are suggested for most Americans. They do not apply to people who need special diets because of disease or conditions that interfere with normal nutrition. Such people may require special instruction from a health professional with expertise in nutritional counseling, such as a registered dietitian, in consultation with their own physicians. These USDA/DHHS guidelines[12] are:

- Eat a variety of foods;
- Maintain desirable weight;
- Avoid too much fat, saturated fatty acids, and cholesterol;
- Eat foods with adequate starch and fiber;
- Avoid too much sugar;
- Avoid too much sodium; and
- If you drink alcoholic beverages, do so in moderation.

The first two guidelines form the framework of a good diet—eating a variety of foods to ensure getting the essential nutrients, and eating only enough calories to maintain desirable weight. The next five guidelines describe special characteristics of a good diet—avoiding too much fat, saturated fatty acids, and cholesterol; getting adequate starch and fiber; and avoiding too much sugar, sodium, and alcohol.

This report focuses on the two guidelines that are particularly related to blood cholesterol and the prevention of CHD: Avoid too much fat, saturated fatty acids, and cholesterol; and maintain desirable weight.

The guidelines also set forth the principle that reductions in energy derived from fat should be offset by increased consumption of foods containing complex carbohydrates such as starch and fiber. The Population Panel recommends that between 50 percent and 60 percent of calories come from carbohydrates.

Several valuable documents have been developed to assist people to follow the Dietary Guidelines for Americans. These documents can also be used to follow the recommendations in this report. These include four new booklets to help consumers put the Dietary Guidelines into practice,[19] and *Dietary Guidelines and Your Diet,*[20] which includes "A Pattern for Daily Food Choices." The pattern suggests daily servings from the major food groups.[21] Valuable information is also contained in booklets and brochures available from the National Institutes of Health and the American Heart Association.

RECOMMENDATION B. 1: The panel recommends that healthy Americans, both adults and children, select, prepare, and consume foods that contain lower amounts of saturated fatty acids, total fat, and cholesterol.

The following specific practices can help Americans, both adults and children, lower blood cholesterol levels. These food choices and preparation methods form the framework of a good diet. Eating a variety of foods to get essential nutrients and only enough calories to maintain desirable weight is also important. These eating patterns are fully compatible with cultural and ethnic considerations and with personal preferences for good food.

- **Fruits, vegetables, whole grain products, legumes such as beans and peas.** These products contain complex carbohydrates, fiber, and minimal amounts of saturated fatty acids. Accordingly, it is recommended that healthy Americans:

  Eat a greater quantity and variety of fruits, vegetables, breads, cereals, and legumes. These choices will help to meet nutritional needs for minerals, vitamins, dietary fiber (including soluble fiber), and complex carbohydrates, and to replace calories from fat.

- **Low-fat dairy products**. Dairy products constitute a major source of fat and saturated fatty acids in the average American diet. For this reason, it is recommended that healthy Americans:

  Eat more low-fat dairy products such as skim or low-fat milk and skim or low-fat milk products. Choose them, in place of regular whole milk, cheese, cream, ice cream, butter, or other butterfat-rich products, to help meet nutritional needs for calcium, protein, and vitamin D (skim milk is an excellent source of calcium with minimal accompanying fat).

- **Low-fat meat, poultry, fish**. Red meats, both in surface fat and fat within the meat tissue, provide a substantial proportion of fat and saturated fatty acids for the average American. Choosing lean cuts of beef, pork, veal, or lamb, removing surface fat, and substituting fish or skinless poultry can significantly reduce saturated fatty acid intake. Thus, the panel recommends that healthy Americans:

  Eat moderate amounts (e.g., about 6 ounces per day, cooked) of trimmed, lean red meat, poultry without skin, or fish in place of choices high in saturated fatty acids. These choices will help meet nutritional needs for protein, iron, and zinc.

  Cholesterol is found only in products from animals.

  - Organ meats—liver, sweetbreads, kidneys, and brain—are very rich in cholesterol, and consumption should be limited.

- **Eggs**. Egg yolk serves as an important source of dietary cholesterol for most Americans. An average large egg yolk contains 213 mg of cholesterol. These facts lead the panel to recommend that healthy Americans:

Eat egg yolks only in moderation. Egg whites do not contain cholesterol and they can be eaten often.

- **Fats and oils**. All vegetable products are free of cholesterol. Many oils come from vegetable products. Most contain large proportions of monounsaturated and polyunsaturated fatty acids and small proportions of saturated fatty acids. Tropical oils such as palm kernel oil, coconut oil, and palm oil contain relatively large proportions of saturated fatty acids. Accordingly, the panel recommends that healthy Americans:

  Use oils, margarines, and shortenings with vegetable oils containing primarily unsaturated fatty acids instead of saturated fatty acids. Consumption of tropical oils, especially the most highly saturated palm kernel and coconut oils, should be minimized.

- **Baked goods**. Most commercially prepared baked goods contain ingredients composed of saturated oils or fats; many contain egg yolks. For these reasons, the panel recommends that healthy Americans:

  Choose prepared baked goods that have been made with unsaturated vegetable oils and, at most, small amounts of egg. Breads and most rolls are low in saturated fatty acids and cholesterol while other commercial products such as croissants, cakes, biscuits, doughnuts, and muffins are often high in saturated fatty acids.

- **Convenience Foods**. Increasingly, Americans are consuming meals, entrees, mixes, and other packaged foods that are conveniently reconstituted and/or reheated. While many of these products have constituents containing large proportions of saturated fatty acid, many do not. Accordingly, it is recommended that healthy Americans:

  Choose "convenience foods" guided by low saturated fatty acid, total fat, and cholesterol content as well as by cost considerations.

- **Food labels**. Increasingly, valuable information is presented on food labels concerning amounts and sources of saturated fatty acids, fat, cholesterol, and other nutrients. For this reason, the panel recommends that all healthy Americans:

  Know your food by reading food labels, including both ingredient and nutrition information. Choose more often foods with lower amounts or proportions of saturated fatty acids and/or fat and of cholesterol. Saturated fatty acids and cholesterol are often found together in foods. Saturated fatty acids are provided primarily by animal products such as the fat in meat, butter, whole milk, cream, cheese, and ice cream. These foods are also major sources of cholesterol.

- **Food preparation**. Preparation of food can significantly increase the proportion of calories from all fat and from saturated fatty acids. For this reason, it is recommended that healthy Americans:

  Keep use of fats to a minimum when preparing foods. Use the smallest amount possible of fat and fatty foods as ingredients. Cook foods in ways that minimize or eliminate fat content, such as broiling, boiling, steaming, microwaving, or baking, instead of frying in fats;

  Use smaller amounts of ingredients high in saturated fatty acids. Where fats are necessary, substitute fats high in unsaturated fatty acids for fats high in saturated fatty acids; substitute equal or smaller amounts of unsaturated margarine or oil for butter;

  Use low-fat alternatives in food preparation. For example, substitute 1%, skim, or reconstituted nonfat dry milk for whole milk or higher fat choices. Use low-fat yogurt, buttermilk, or evaporated skim milk in place of sour cream, cream, or cream cheese.

- **Eating out**. Americans, on average, derive a significant portion of their nutrition outside of the home. Thus, the panel recommends that healthy Americans:

In restaurants, select menu items that are low in saturated fatty acids, total fat, and cholesterol as well as cooked foods that are baked, boiled, or broiled without fat. Ask for lean cuts of meat trimmed of excess fat. Request that sauces and salad dressings be served in separate containers if high in fat and use them sparingly. Ask for information on ingredients and preparation of restaurant food. When eating out and on other social occasions, choose foods with less fat—e.g., larger amounts of vegetables, fruits, and grain products, and smaller amounts of foods that are likely to be high in saturated fatty acids.

- Fast foods.

  While increasing numbers of "fast food" outlets have choices in which low proportions of calories are derived from saturated fatty acids, many others contain large amounts of saturated fatty acids and cholesterol. For these reasons, the panel recommends that healthy Americans:

  Ask for information on the fat and saturated fatty acid content of "fast food" selections and choose foods that are nutritious and low in saturated fatty acids, total fat, and cholesterol.

- **Overly simple solutions**. An habitual pattern of eating that is consistently low in saturated fatty acids, total fat, and cholesterol is recommended. Focusing solely on the elimination of a single food such as egg yolks, however, is not necessary and will not, by itself, achieve sufficient blood cholesterol lowering. As indicated earlier, skim or low-fat dairy products and small portions of trimmed, lean red meat are desirable and nutritionally valuable. These foods and eggs in moderation can all be part of a blood cholesterol-lowering eating pattern. The panel therefore recommends that all healthy Americans:

Recognize that no single food or supplement is the answer to achieving a desirable blood cholesterol level.

- Fish oil supplementation will not lower blood cholesterol levels in most people. However, consumption of fish, a food low in saturated fatty acids, instead of foods that are higher in saturated fatty acids, is desirable.

- Dietary fiber supplements are not a panacea for blood cholesterol problems. Foods rich in soluble dietary fiber are, however, a useful addition to a low saturated fatty acid, low-fat, and low-cholesterol eating pattern.

- Alcohol is not recommended as a means of preventing CHD because it has not been shown to be effective and because of the potentially harmful effects of excessive alcohol intake.

RECOMMENDATION B. 2: The panel urges the public to recognize that an elevated blood cholesterol level is one of the important modifiable CHD risk factors together with smoking, high blood pressure, excess body weight, and physical inactivity.

The recommended eating patterns should be considered part of a personal health program which also emphasizes the following risk factors for coronary heart disease.

- **Smoking, high blood pressure.** Being a nonsmoker, and preventing and controlling high blood pressure are both important factors in prevention of CHD.

- **Excess body weight.** It is important for all healthy Americans to achieve and maintain desirable body weight by limiting energy (calorie) intake and by engaging in regular moderate exercise. Maintenance of desirable body weight is a useful means of controlling blood cholesterol.

- **Physical inactivity.** Apart from beneficial effects on body weight and on blood pressure, regular exercise throughout life is associated with a lower risk of CHD.

C. Healthy Children and Adolescents

RECOMMENDATION C. 1: The panel recommends that healthy children follow the recommended eating patterns that are lower in saturated fatty acids, total fat, and cholesterol as they begin to eat with the family, usually at 2 years of age or older.

The Population Panel has reviewed evidence concerning benefits and safety for children and adolescents of an eating pattern that is lower in saturated fatty acids, total fat, and cholesterol than the usual American diet. The panel also recognizes that the caloric and nutrient needs of children are critical for supporting growth and development. Since eating habits developed during childhood can influence lifetime practices, the panel urges prudent movement to the recommended eating pattern. Healthy children should be allowed to share in family food choices, recipes, and menus. Adolescents are particularly encouraged to maintain a low saturated fatty acid, low total fat, and low-cholesterol eating pattern.

Infants and children under the age of 2 years have dietary requirements different from those of older people. Infants whose diet is primarily mother's milk or formula often appropriately consume 40 percent or more of calories from fat. The Population Panel recommends that this well-established pattern of infant nutrition be encouraged. Care must be taken to ensure sufficient energy and nutrient intake to meet the needs of the growing child. A forthcoming report from the Expert Panel on Blood Cholesterol Levels in Children and Adolescents will deal with strategies for detection, evaluation, and treatment of children and adolescents at high risk of later CHD because of high blood cholesterol levels and will provide additional details about implementation of strategies for encouraging desirable eating patterns for healthy children.

D. Special Groups

RECOMMENDATION D. 1: The panel recommends that particular attention be paid to nutritional concerns of special segments of the population.

The eating patterns of specific groups of Americans should be considered. Population groups with special dietary concerns include women, older Americans, cultural and ethnic groups, low-income groups and individuals with special nutritional needs.

- **Women**. Women, like men, can and should adopt the recommended eating patterns that are lower in saturated fatty acids, total fat, and cholesterol as a means of reducing their likelihood of disease. The special needs of women for nutrients such as calcium, iron, and zinc can be met within these eating patterns.

- **The elderly**. For most individuals 65 years of age and older, the recommended eating patterns that are lower in saturated fatty acids, total fat, and cholesterol, that help maintain desirable body weight, and that are nutritionally adequate, are appropriate. Special attention, possibly including nutrition guidance from a qualified health professional with special nutrition expertise, may be needed for some elderly people who have special nutritional needs or who are limited in their ability to procure, prepare, or consume food.

- **Cultural groups**. Foods and menus should accommodate cultural, regional, and ethnic preferences, while incorporating the recommended intakes of saturated fatty acid, total fat, and cholesterol.

- **Low-income groups**. Low-cost foods meeting the recommended eating patterns are available; individuals of lesser economic means can and should select these to improve their diets and share in better health.

- **People with special nutritional needs.** Health professionals should be attentive to individuals who have special nutritional needs, such as pregnant or lactating women, patients with food allergies, and many patients with diabetes mellitus, or chronic kidney, heart, or liver disease, who often need counseling from a health professional with special nutritional expertise.

E. Health Professionals

RECOMMENDATION E. 1: The panel recommends that health professionals advise patients and the public to attain the recommended eating patterns.

Physicians, nurses, registered dietitians, nutrition and health educators, pharmacists, and other health professionals have the opportunity to be important influences on patients and the public concerning the attainment of the panel's recommendations. Health professionals should both practice and advocate the recommended eating patterns. Initial and continuing nutrition education for health professionals should emphasize the panel's recommendations. Health professionals, as needed, should obtain assistance from others with special nutrition counseling expertise to facilitate achievement by patients and the public of the recommended eating patterns. In particular, health professionals should:

- Ensure that adult patients have their blood cholesterol measured accurately and that the meaning of the results is clearly explained in keeping with ATP guidelines;
- Encourage and counsel their patients, as members of the public, to follow eating patterns that are low in saturated fatty acids, total fat, and cholesterol but that are nutritionally adequate;
- Support the use of more effective communication approaches for populations such as low-income, low-education, and minority groups, encouraging them to adopt these eating patterns;
- Provide shopping and food preparation guidelines that include economical food alternatives that are lower in saturated fatty acids, total fat, and cholesterol and that incorporate cultural, ethnic, and personal food preferences;
- Develop simple dietary self-assessment and self-monitoring techniques to help individuals evaluate and monitor their own eating patterns;
- Work with industry, government, and voluntary groups to improve, expand, and simplify consumer-oriented nutrition information, advertising, and labeling;
- Encourage health departments, hospitals, and clinics to provide nutrition education and services consistent with the nutrient intakes and eating patterns recommended by this panel through efforts such as printed materials, videotapes, and nutrition information services;
- Encourage all medical and other health professional schools to incorporate educational components consonant with the eating patterns and principles recommended by the panel;
- Work with health care agencies such as the medical centers of the Department of Veterans Affairs, the military health care system, private and public hospitals, local neighborhood ambulatory health centers, and other health care delivery organizations, to help patients and staff adopt and follow the eating patterns recommended by the Population Panel;
- Identify individuals with special nutritional needs or eating problems who may benefit from assessment and intervention by a qualified health professional with expertise in nutrition counseling such as a registered dietitian; and
- Serve as role models for the public by adhering to the recommended eating patterns.

F. The Food Industry

What people eat is influenced by many factors. These include both individual factors and the nutritional environment of the Nation. Individual factors include the variety of cultural heritages, economic factors, the availability of food, the taste of food, and knowledge about food. The environment includes a broad array of influences on the food chain. Recognizing the influence of each of these factors on the food that Americans eat, the panel makes the following recommendations concerning the diverse elements in the U.S. food chain.

RECOMMENDATION F. 1: The panel recommends that food producers, manufacturers, and distributors increase the availability of good-tasting foods that are lower in saturated fatty acids, total fat, and cholesterol.

This can be accomplished through the following activities.

- Expand efforts to produce, manufacture, and market animal products lower in saturated fatty acids, total fat, and cholesterol with particular emphasis on leaner cuts of meat and lower fat dairy products;
- Continue efforts by the agriculture industry to produce, manufacture, and market processed plant products containing smaller amounts of saturated fatty acids, and larger amounts of complex carbohydrate and dietary fiber, including soluble fiber;
- Continue development of safe food preservation techniques suitable for products containing unsaturated fatty acids;
- Continue development of methods for mass preparation of food containing lower levels of saturated fatty acids, total fat, and cholesterol;
- Continue development of foods and menus that will help people attain the recommended intakes of saturated fatty acids, total fat, and cholesterol and that will ensure adequate nutrient intakes;
- Encourage food industry and agricultural commodity groups to adopt products, policies, and positions consistent with these recommendations while assuring both continued responsiveness to consumer desires and necessary profitability; and
- Provide professional education materials, and consumer brochures, cookbooks, and recipes, as well as other print and nonprint educational materials indicating ways of practicing the recommended eating patterns.

RECOMMENDATION F. 2: The panel recommends that the food industry participate actively in helping the public attain desirable eating patterns through labeling and advertising activities.

This can be accomplished through the following activities.

- Promote foods that are low in saturated fatty acids, total fat, and cholesterol, working with the advertising industry and the media to ensure that accurate messages reach large segments of the public;
- Provide comprehensible quantitative information on most food labels concerning saturated fatty acids, total fat, cholesterol, and calories to enable consumers to make informed choices;
- Avoid misleading the public through advertising or product labeling (e.g., refrain from promoting the "no cholesterol" claim for plant origin products when these products contain relatively large amounts of fat, especially saturated fatty acids, and avoid making health claims that are not supported by scientific evidence);
- Publicize the good taste and economy as well as the health-promoting characteristics of an eating pattern low in saturated fatty acids, total fat, and cholesterol; and
- Encourage hard-to-reach segments of the population such as low-income, low-education, low-literacy, and minority groups to choose food products that are low in saturated fatty acids, total fat, and cholesterol.

RECOMMENDATION F. 3: The panel recommends that food vendors and other food distribution sites participate actively in the national effort.

This can be accomplished through the following activities.

- Develop and disseminate point-of-purchase information in individual grocery stores, supermarket chains, restaurants, and cafeterias to identify foods low in saturated fatty acids, total fat, and cholesterol;
- Label, promote, and expand the selection of food choices that are lower in saturated fatty acids, total fat, and cholesterol in places providing food to large numbers of people such as institutional food services, restaurants, convenience or "fast food" outlets, schools, worksites, government agencies, hospitals, transportation services, catering services, and other feeding programs;
- Expand the availability and encourage the selection of foods that are lower in saturated fatty acids, total fat, and cholesterol in government-subsidized or -supervised food programs (e.g., school lunch programs); and
- Continue to develop ethnically and culturally appropriate food selections that are lower in saturated fatty acids, total fat, and cholesterol.

RECOMMENDATION F. 4: The panel recommends that the food industry, including food and animal scientists, food technologists, and nutritionists, continue to develop and modify foods to help the public meet the recommended eating patterns.

This can be accomplished through the following activities.

- Intensify research and development of food products that are lower in saturated fatty acids, total fat, and cholesterol;
- Continue development of economically, feasible uses of food processing by-products—such as meat trimmings—containing saturated fatty acids, fats, and cholesterol, other than as human food; and
- Assure that as substitutes for food components such as fat are developed, there is clear evidence of safety for human consumption.

G. Mass Media

RECOMMENDATION G. 1: The panel recommends that the mass media provide information on a lower saturated fatty acid, lower total fat, and lower cholesterol eating pattern.

Through their news, information, entertainment, and advertising policies and programming, the mass media have the ability to influence large numbers of people. As critical channels of communication to the public, the mass media are extremely important allies in the effort to reduce blood cholesterol levels throughout the population.

H. Government

RECOMMENDATION H. 1: The panel recommends that government facilitate attainment of healthful eating patterns by modifying policies and approaches.

Federal, state, and local governments are responsible for many programs and policies related to nutrition and to health. The following governmental activities will help promote cholesterol-lowering eating patterns.

- Increase collaboration among government agencies—e.g., the USDA, the Federal Trade Commission, the Federal Communications Commission, the U.S. Department of Veterans Affairs, and the DHHS including the Food and Drug Administration (FDA), the National Institutes of Health (NIH), and the Centers for

Disease Control—to provide consistent, coordinated nutrition statements and policies emphasizing low saturated fatty acid, low-fat, and low-cholesterol eating patterns;

- Help consumers make informed choices through expansion and standardization of food labeling requirements, especially through collaborative efforts of the FDA and USDA with the food industry, to include on most packaged foods easily comprehensible labels that clearly identify, among other nutritional characteristics, the content of saturated fatty acids, total fat, and cholesterol, as well as total calories;
- Develop, implement, and educate consumers concerning quantitative definitions for such terms as "lean," "low-fat," "low in saturated fatty acids," and "lite";
- Improve labeling of meat to help consumers select lean cuts;
- Increase state and federal government efforts to provide consistent nutrition education and food choice guidance for the public emphasizing the desirability of foods with low-saturated fatty acid, low total-fat and low-cholesterol content; and
- Revise food-related policies to stimulate production and distribution of foods low in saturated fatty acids, total fat, and cholesterol (e.g., the recent encouraging movement toward reduction of saturated fatty acid content of school lunch programs).

I. Educational Systems

RECOMMENDATION I. 1: The panel recommends that all public and private educational systems become active partners by disseminating information about the role of eating patterns in CHD prevention.

Attainment of lower blood cholesterol levels and reduced risk of CHD will be enhanced if educational programs at all levels—elementary through high school grades, vocational schools, colleges, universities, and medical schools—increase nutrition education. Accordingly, the panel recommends a focus on low saturated fatty acid, low-fat, and low-cholesterol eating patterns in the following nutrition education materials and activities:

- Curricula in health education, science, and home economics classes as appropriate for kindergarten through 12th grades;
- Curricula of culinary arts education programs emphasizing purchase, preparation, and presentation of foods and menus low in saturated fatty acids, total fat, and cholesterol;
- Curricula of university food science departments, including consideration of how advances in molecular biology, genetic, and production techniques can facilitate development of new food products with reduced saturated fatty acid, total fat, and cholesterol content;
- Curricula of college and university undergraduate programs, especially biological science, health, nutrition, and physical education; and
- Curricula of all pre- and postdoctoral physician and other health professional education programs (e.g., for nurses, dietitians, and pharmacists).

RECOMMENDATION I. 2: The panel recommends that existing nutrition education programs expand and be compatible with the recommended eating patterns, and that messages from major organizations concerning health-promoting eating patterns be consistent.

Many organizations already offer nutrition education programs to help Americans reduce cholesterol levels or meet other dietary goals. Examples are programs offered or supported by voluntary organizations (e.g., American Heart

Association, American Cancer Society) or by federal, state, or local government agencies. Education programs offered through the network of USDA's Extension Service can effectively communicate nutrition information to targeted audiences. Divergent messages can confuse the public; consistent, coherent information is needed.

J. Measurement of Blood Cholesterol

RECOMMENDATION J. 1: The panel recommends that public screening for blood cholesterol be undertaken only under conditions that ensure adherence to high quality standards.

Measurement of blood cholesterol serves to augment awareness of elevated blood cholesterol as a personal health problem. Recognizing the vital importance of cholesterol awareness, the NCEP has promoted a broadly based public education program—"Know Your Cholesterol Number"—to encourage all adult Americans to know their blood cholesterol level.

The ATP recommended that all American adults have their blood cholesterol measured at least once every 5 years. Cholesterol measurements should meet the accuracy and precision standards of the Laboratory Standardization Panel. The usual approach to detecting high blood cholesterol is through the physician's office. In addition, public screening provides the possibility of detecting large numbers of individuals with high blood cholesterol. However, the reliability of cholesterol measurements, the education of participants, and proper referral and followup in public screening programs all need special attention.

Only under specific conditions should general public screening for blood cholesterol be used to supplement screening in the health care setting. The NHLBI Workshop Regarding Public Screening for Measuring Blood Cholesterol[22] recommended the following criteria for public screening.

- Use recruitment approaches that attract all adult segments of the community and develop special approaches for harder-to-reach target groups. These include men, younger adults, low-income or low-education groups, and minorities;
- Evaluate the performance of each cholesterol analyzer before conducting a public cholesterol screening, and document ongoing internal and external quality control procedures, to ensure that cholesterol measurements are precise and accurate, and that they meet the standards of the Laboratory Standardization Panel of the NCEP;
- Ensure that public screening programs are managed by staff trained and supervised by appropriate health professionals and are supported by a qualified health institution and that the quality of all aspects of screening programs is evaluated;
- Provide cholesterol screening at a reasonable cost to the participants, with moderation of the cost for certain low-income group participants as necessary;
- Provide reliable verbal and printed educational information about cholesterol levels from knowledgeable staff, so that individuals understand the meaning and limitations of a single cholesterol measurement, receive information on dietary practices to lower cholesterol, and are given clear instructions with respect to their own medical followup;
- Recommend referrals on the basis of the NCEP guidelines shown in table 4, page 30; and
- Emphasize that screening is not a substitute for health care and cannot replace medical monitoring of blood cholesterol levels of individuals already under treatment.

Recommendations for screening children and adolescents and for managing younger people with elevated cholesterol levels will emerge later from the NCEP's Expert Panel on Blood Cholesterol Levels in Children and Adolescents.

Table 4

Guidelines for Referral of People Based on Their Blood Cholesterol Level

Blood Total Cholesterol Level	**Recommended Action**
Greater than or equal to 240 mg/dL (High)	Refer to physician for followup.[*]
Between 200 and 239 mg/dL (Borderline-High)	Refer to physician for followup[*] if history of coronary heart disease (CHD) or if two or more other CHD risk factors (excluding HDL-cholesterol) detected on interview (see table 2, page 9). If no reported history of CHD or less than two other risk factors, refer to physician within 1 year for repeat cholesterol measurement.
Less than 200 mg/dL (Desirable)	Recommend a repeat blood cholesterol followup in 5 years.

[*] Individuals should be seen by their physicians within 2 months.

Source: NCEP ATP, January 1988[3]

K. Research and Surveillance

RECOMMENDATION K. 1: The panel recommends that research and evaluation on the relationships between food and health continue and that the panel's recommendations be reconsidered in a timely manner as new scientific information becomes available.

The following specific activities should be undertaken:

- Continue efforts to clarify even further the role and the interactions of dietary components and nutrients, and blood lipids, atherosclerosis, and CHD;
- Improve analytical methods for food composition and continue development and application of the National Nutrient Databank of commodity and commercial products, including brand name information as appropriate to provide reliable information about nutrient composition of foods consumed by Americans;
- Collect and evaluate data, individual and societal, pertaining to dietary change along with evaluations of effects on CHD and other health status indicators. These data should include information on food production, processing, and consumption patterns and costs; population levels of blood cholesterol; and trends in cause-specific morbidity and mortality. Information concerning the efficacy of reaching all segments of the population should be

collected at the national, state, regional, and local levels. Many of these databases are ongoing through the work of government agencies—e.g., NHANES surveys; National Center for Health Statistics (NCHS) mortality statistics; FDA/NHLBI consumer and NHLBI health professional surveys; FDA total diet surveys; USDA nationwide food consumption surveys; state-based behavioral risk factor surveys—and should be continued or improved, while other data needs will require development of new monitoring systems;

- Expand research and development related to the reduction or alteration of fat content in foods of animal origin as recommended in the National Research Council report, *Designing Foods*;[11] and
- Continue to identify, develop, and evaluate effective nutrition education and communication methods.

V. Scientific Evidence for Recommendations Affecting the General Public

A. Diet Patterns, Blood Cholesterol, and Health

1. Introduction

This review examines the scientific literature concerning the etiologic factors for increased blood cholesterol and CHD. It presents the diversity of observations and interpretations that link diet, blood cholesterol, and CHD. The preponderance of the data in the literature indicate that habitual diet directly influences the level of blood cholesterol, and that in turn, elevated levels of blood cholesterol result in high rates of CHD.

2. Clinical, Laboratory, and Metabolic Research

a. Lipids and Lipoproteins

Lipids circulate in blood plasma in association with certain specific proteins (apolipoproteins) as large, macromolecular complexes called plasma lipoproteins. The lipoproteins are classified as chylomicrons, very low density lipoprotein (VLDL), low density lipoprotein (LDL), and high density lipoprotein (HDL). Lipoproteins are lighter than the other plasma proteins because of their high lipid content. In turn, their weight or density varies from high to very low largely as a function of their relative contents of protein and various lipids.

The primary function of plasma lipoproteins is lipid transport. The major lipid transported in lipoproteins—triglyceride—is insoluble, yet up to several hundred grams must be transported through the blood daily. Cholesterol is the other major lipid transported in lipoproteins. It is not used for energy production, as are triglycerides, but is the precursor of steroid hormones and bile acids and serves as an important structural component of cellular membranes. In higher animals, it is transported in plasma predominantly as cholesteryl esters (CE). The transport of cholesterol in lipoproteins permits specific targeting of cholesterol to tissues that require it for structural purposes or to make metabolic products.

Two of the lipoprotein classes, chylomicrons and VLDL, contain large quantities of triglyceride and represent, respectively, the transport form of exogenous (dietary) and endogenous triglyceride. Chylomicrons normally are not present in post-absorptive plasma after an overnight fast. The VLDL normally contains 10-15 percent of the total plasma cholesterol. LDL contains cholesterol as its major component and normally contains most (60-70 percent) of the plasma cholesterol. HDL is comprised of approximately half protein and half lipid, and usually contains 20-30 percent of the total plasma cholesterol.

Each lipoprotein class is heterogeneous with respect to its protein constituents. At least nine distinct apolipoproteins have been separated and described. Most investigators group the apolipoproteins into five families (designated apoA, apoB, apoC, apoD, and apoE) on the basis of their chemical, immunologic, and metabolic characteristics.[23,24] In addition, a distinct LDL-related particle called lipoprotein (a) consists of an unusual apolipoprotein, designated apo (a), attached to an LDL particle. Elevated levels of lipoprotein (a) have been associated with an increased risk of atherosclerotic disease.[25,26]

b. Atherogenesis

The strong relationship between high levels of plasma LDL-cholesterol and atherosclerosis in man has stimulated in vitro studies dealing with the cellular and molecular mechanisms of atherogenesis. These studies have sought to determine how LDL-cholesterol and other "atherogenic" lipoproteins could promote the formation of foam cells and atherosclerotic lesions. The results of such in vitro studies have, in turn, supported the human and animal data that suggest a fundamental etiologic role for LDL and related lipoproteins in the cause of atherosclerosis.

A prominent and early feature of atherosclerotic lesions is the CE-filled macrophage, or foam cell. To understand the mechanism(s) of foam cell formation, investigators have studied the interactions of plasma lipoproteins with various types of cultured macrophages. Native LDL does not lead to CE accumulation in most types of macrophages.[27] This is due mainly to the fact that production of the LDL receptor on these cells is down-regulated by small amounts of excess cellular cholesterol, thus preventing a large influx of LDL-cholesterol.[28] However, various modified forms of LDL such as acetyl-LDL, oxidized LDL, and malondialdehyde-LDL do lead to marked accumulation of CE in cultured macrophages. These modified forms of LDL enter the cell by a receptor (called the "scavenger" receptor) that is distinct from the LDL receptor and that is not subject to down-regulation.[27,29] Thus, cellular influx of these modified forms of LDL continues over time at a high rate, leading to significant CE accumulation.

These in vitro observations have spawned a widespread search for evidence of LDL modification in vivo. Several investigators have reported the occurrence of various kinds of modified LDL in atherosclerotic arteries.[30-33] A potential role for native LDL in foam cell formation has also been suggested by the finding that a particular type of cultured macrophage has a poorly down-regulated LDL receptor and accumulates large amounts of CE in the presence of native LDL.[34]

Foam cell formation has also been demonstrated in vitro by lipoproteins other than modified and native forms of LDL. One such lipoprotein, called β-VLDL, is a cholesterol-enriched lipoprotein found in the genetic disease, familial dysbetalipoproteinemia. β-VLDL causes massive deposition of cholesteryl ester in macrophages in vitro.[27,35] The receptor by which β-VLDL enters the macrophage demonstrates poor down-regulation.[35] Chylomicron remnants, which may occur in humans, appear to interact with macrophages in a manner similar to that of β-VLDL and result in some degree of CE accumulation, as well as a large amount of triglyceride accumulation.[36] These and other data have led some investigators to suggest that some postprandial lipoproteins, particularly cholesterol-enriched lipoprotein particles and remnants, may be atherogenic in man.

Research on lipid-rich monocytes, or foam cells, represents an active area of in vitro investigation exploring the relationship between LDL-cholesterol and atherogenesis. However, other approaches to the exploration of this relationship have also been attempted.[37] For instance, LDL, either native or oxidized, has been demonstrated to cause endothelial cell injury in vitro. Endothelial injury, in turn, has been theorized to be a potentially important initiating event in atherogenesis, leading to platelet adherence and release of growth factors. In addition, LDL-cholesterol has been reported to directly augment platelet aggregation and stimulate the growth of smooth muscle cells, two important features of the atherosclerotic lesion. It is clear, moreover, that a variety of cellular and vessel wall factors, not yet well-defined, are also important in atherogenesis. Ross[38] has reviewed the potential roles of growth factors, of endothelial injury, and of the different arterial wall cells in the pathogenesis of atherosclerosis.

In summary, there have been numerous in vitro studies demonstrating the ability of LDL and related lipoproteins to cause the formation of foam cells and other elements of the atherosclerotic lesion. Although other metabolic, cellular, and vessel wall factors important in atherogenesis exist and remain to be better defined, the in vitro data currently available strongly suggest an important causative role for LDL-cholesterol and related lipoproteins in atherogenesis.

c. Experimental Studies in Animals

Studies in laboratory animals have clearly shown that atherosclerosis can be induced by diet. The first studies of the relationship of diet to atherosclerosis were initiated in the early 1900's. It was not until the 1950's that investigators began to study in animals the effects of fat and different fatty acids.[39,40] Atherosclerosis was first produced experimentally in rabbits by feeding pure cholesterol in 1913.[41] Certain species—i.e., rabbits, a number of avian varieties, pigs, and nonhuman primates—are especially susceptible to severe hypercholesterolemia induced by dietary cholesterol.[42-47] When hypercholesterolemia is maintained for long periods, these animals develop advanced atherosclerotic lesions, similar to those found in humans, and eventually myocardial infarction occurs.[48,49] The close link between dietary cholesterol and atherosclerosis in various animal species,[50] including nonhuman primates, underlies one argument for the importance of dietary cholesterol as an atherogenic factor in humans.

Variability exists among different animals in their susceptibility to atherosclerosis induced by dietary cholesterol. Some species such as rats and dogs do not develop significant hypercholesterolemia or atherosclerosis when fed large amounts of dietary cholesterol.[51,52] Although these animals absorb a high proportion of excess dietary cholesterol, they can rapidly degrade this excess into bile acids, thereby protecting themselves from developing hypercholesterolemia and atherosclerosis.[51-53] These species can develop hypercholesterolemia when their normal ability to dispose of excess dietary cholesterol is blocked by induction of hypothyroidism.[54]

Jokinen et al.[55] have recently reviewed the advantages and disadvantages of various primates as models for dietary-cholesterol-induced atherosclerosis. The amount of cholesterol required to induce atherosclerosis is variable between different species. The administration of dietary cholesterol to susceptible species produces changes in plasma lipoproteins that are similar, or at least analogous, to those associated with enhanced atherogenesis in humans. In nonhuman primates, high-cholesterol intakes cause LDL-cholesterol to rise to levels that rapidly induce atherosclerosis; furthermore, in the same animals, low levels of HDL are inversely related to atherosclerosis.[56,57] In fact, approximately 50 percent of the variance in atherosclerosis in primates can be explained by differences in levels of LDL and HDL.[56-58] Moreover, dietary cholesterol modifies the types of LDL particles present in plasma of some types; for example, Rudel et al.[59] reported that feeding excess cholesterol to primates induces formation of cholesterol-enriched LDL particles that are highly atherogenic.

Dietary cholesterol theoretically could increase LDL-cholesterol levels in laboratory animals by multiple mechanisms, e.g., increased production of cholesterol-enriched lipoproteins and inhibition of clearance of LDL from the circulation. Studies in tissue culture indicate that increasing the cellular content of cholesterol suppresses LDL receptor synthesis and activity.[29] It has been shown in many species that raising dietary cholesterol will cause an increase in hepatic cholesterol content. Spady and Dietschy[60] demonstrated that high cholesterol intakes suppress LDL-receptor-mediated uptake of LDL by hamster liver. Thus, suppression of LDL-receptor activity is probably a major mechanism whereby dietary cholesterol raises LDL-cholesterol levels in plasma.

Different animal species develop different patterns of hyperlipidemia upon feeding of high-cholesterol diets. For example, rabbits show greater increases in VLDL-cholesterol than in LDL-cholesterol levels. Some primates, in contrast, develop abnormally large, cholesterol-rich LDL in preference to increased VLDL-cholesterol.[59] In contrast to both of these species, humans normally show increases of blood cholesterol almost exclusively in normal-sized LDL particles.[61] The underlying mechanism of hyperlipidemia, i.e., reduced LDL-receptor activity, may nonetheless be similar in all these species; only the pattern of response in plasma lipoproteins may be species specific. Still another mechanism for hypercholesterolemia in different species could be replacement of triglycerides by cholesteryl ester in newly secreted lipoproteins. This effect has been noted in lipoproteins secreted by perfused livers of cholesterol-fed animals,[62] and it could contribute to the cholesterol-rich VLDL (β-VLDL) noted in rabbits and hypothyroid dogs. Thus excess dietary cholesterol may raise the plasma cholesterol level by more than one mechanism.

One theory holds that postprandial lipoproteins play an important role in atherosclerosis.[63] According to this view, dietary cholesterol transported in chylomicrons and chylomicron remnants is directly atherogenic independent of its effects on fasting levels of LDL-cholesterol. Research in laboratory animals provides a unique opportunity to evaluate this hypothesis. Only a few studies have actually examined postprandial lipoproteins in laboratory animals.[64,65] Studies in primates have shown that cholesterol-rich, postprandial lipoproteins, resulting from high intakes of dietary cholesterol, may produce atherosclerosis. Future research in various animal species may provide new insights into the role of cholesterol-rich, postprandial lipoproteins in atherogenesis.

Not only will the feeding of high levels of dietary cholesterol increase the plasma cholesterol level and induce atherosclerosis in many animals, but removal of cholesterol from the diet will lower plasma cholesterol levels and cause regression of atherosclerotic lesions.[66-69] This finding adds further support for a role of dietary cholesterol in the genesis of atherosclerosis.

Nonhuman primates, as well as other animal species, are affected by the types of fatty acids in the diet, especially during feeding of high-cholesterol diets.[70] In primates, when the diet is rich in cholesterol, saturated fatty acids raise blood cholesterol levels, whereas unsaturated fatty acids lower them.[71-73] In baboons, selective breeding can influence the response to saturated fatty acids.[74] In some studies polyunsaturated fatty acids appeared to lower cholesterol levels more than monounsaturated fatty acids, but the studies by Spady and Dietschy[75] in hamsters suggested that in this species the two types of unsaturates have about the same effect on LDL-cholesterol levels.

Laboratory animals should be good models for investigations of mechanisms whereby SFA raise the cholesterol level. Studies by Spady and Dietschy[60,75] showed in hamsters fed with cholesterol that SFA suppress hepatic LDL-receptor activity. Fox et al.[76] demonstrated further that SFA reduce levels of messenger RNA for synthesis of LDL receptors. These studies point to a suppression of LDL receptors as a primary mechanism whereby SFA raise LDL-cholesterol levels in laboratory animals.

d. Human Studies

Metabolic ward studies and other research with carefully controlled diets provide important insights into the effects of individual dietary components on the plasma lipids and lipoproteins in humans. These investigations have lent support to other work indicating that at least three factors raise the blood cholesterol level: an increased intake of SFA, an increased intake of cholesterol, and the presence of obesity. Among primate species, humans appear to be less susceptible to the hypercholesterolemic effects of dietary cholesterol than most other primates (see section c, Experimental Studies in Animals). In contrast, humans seem to be more sensitive to the influence of different dietary fatty acids than are most species of nonhuman primates. The following summarizes the effects of different nutrients on lipoprotein levels as revealed by studies on the metabolic ward or under tightly controlled experimental conditions.

The major action of SFA is to raise the LDL-cholesterol concentration.[77-84] Several different types of SFA are present in the diet, and these produce different responses in LDL-cholesterol levels. Dietary SFA generally range from 4 to 18 carbon atoms (C4, C6, C8, C10, C12, C14, C16, and C18). Several investigators[85-87] have reported that the C6-C10 SFA do not raise the blood total cholesterol level. The major cholesterol-raising SFA are the C14 and C16 acids.[78,79] Hegsted et al.[78] reported that lauric acid (C12) is less hypercholesterolemic than myristic acid (C14) and palmitic acid (C16). In contrast, Keys et al.[88] reported that C12-C16 SFA are similar in their cholesterol-raising properties. Finally, it was postulated more than two decades ago that stearic acid (C18) does not raise blood cholesterol levels;[77,78,88] recent studies support this claim and have further shown that stearic acid, at least when occupying the 1 and 3 positions of the triglyceride molecule, does not increase LDL-cholesterol concentrations.[84] Moreover, since hydrogenation of vegetable oils that are rich in oleic acid and linoleic acid produces stearic acid as one of its end products, many oils subjected to this process may not yield a product that will raise blood cholesterol levels. Although further research is needed to identify the particular effects of individual fatty acid constituents, as well as their effects when combined in actual dietary settings, the fact that most SFA in the American diet raise blood cholesterol level is firmly established.

How then do SFA raise LDL-cholesterol levels? The best available answer, based on animal studies, is that high intakes of SFA suppress the synthesis of LDL receptors[60,75] leading to increased LDL levels. The final word on this question, however, has yet to be written; SFA may have more than one action on cholesterol and lipoprotein metabolism.

The major PUFA in the diet is linoleic acid, which is an omega-6 fatty acid. For many years linoleic acid held a special place among other nutrients for its putative "cholesterol-lowering" action.[77,78,89-91] A favorite question was, "How do polyunsaturated fatty acids lower the cholesterol level?" A variety of mechanisms have been proposed, e.g., increasing the excretion of cholesterol and bile acids from the body,[92-95] redistributing cholesterol from the plasma into tissues,[96,97] altering the structure of lipoproteins so that they can hold less cholesterol,[98] increasing cholesteryl ester polyunsaturates, and other mechanisms.[99] More recent work suggests that when PUFA replaces SFA in the diet, the major action of linoleic acid is to increase clearance of LDL from plasma through an increased activity of LDL receptors. Although the latter mechanism is probably a major action of linoleic acid, this fatty acid may have multiple effects on lipoprotein metabolism, as suggested by previous studies.

When linoleic acid replaces SFA in the diet, the major response is a lowering of LDL-cholesterol levels.[100,101] Keys et al.[90] and Hegsted et al.[78] proposed that linoleic acid has a unique action to reduce total cholesterol more than other nutrients (i.e., MUFA and carbohydrates); it has since been widely assumed that this effect extends to LDL-cholesterol levels as well. A recent study[80] showed that replacement of SFA by either linoleic acid or oleic acid caused significant reductions in total cholesterol and LDL-cholesterol levels; no significant differences were seen between linoleic and oleic acids. Similar results have been found by Mensink and Katan.[102]

High intakes of linoleic acid in some people can reduce levels of VLDL-triglyceride, VLDL-cholesterol, and HDL-cholesterol.[95,101,103,104] These reductions are greater than those seen for monounsaturates[80] and thus may partly explain enhanced total-cholesterol lowering by linoleic acid compared with oleic acid, as reported by Keys et al.[90] and Hegsted et al.[78] On the other hand, the lowering of VLDL and HDL is pronounced only at high intakes of linoleic acid; at the more moderate intakes of typical diets, a significant reduction in VLDL and HDL likewise has been difficult to detect.[105,106]

The omega-3 PUFA, eicosapentaenoic acid (EPA) (20:5, $\omega - 3$) and docosahexaenoic acid (DHA) (22:6, $\omega - 3$), are found mainly in fish oils. These fatty acids have unique effects on lipid metabolism. Their most striking action is to lower plasma triglyceride.[107,108] This action is a result of their inhibition of the production of VLDL-triglyceride. Seemingly, omega-3 polyunsaturates act in the liver to prevent the incorporation of other fatty acids into triglycerides. As a result, total secretion of VLDL-triglyceride is reduced.[109,110]

The omega-3 PUFA probably do not have an independent effect by which they lower LDL levels. Like other unsaturates, when omega-3 PUFA replace SFA in the diet, the LDL-cholesterol level will fall.[111] This response probably is, at least in part, the result of removing SFA from the diet. Supplementation of the diet with omega-3 polyunsaturates, without altering the intake of saturated fatty acids, does not cause a lowering of the LDL-cholesterol level.[112,113] In hypertriglyceridemic patients who are treated with supplements of omega-3 fatty acids, the decline in plasma triglycerides frequently is accompanied by an increase in LDL-cholesterol concentration.[110]

The actions of omega-3 PUFA on HDL-cholesterol levels are variable. The decrease in triglyceride levels in hypertriglyceridemic patients can be associated with an increase in HDL-cholesterol levels. In the absence of hypertriglyceridemia, however, replacement of SFA by omega-3 PUFA can cause a fall in HDL-cholesterol levels[110] as also occurs when large amounts of omega-6 fatty acids are substituted for saturated fatty acids. Finally, supplementation of the diet with fish oils without other changes in the diet usually has little or no effect on HDL concentrations.

The major fatty acid in most diets is oleic acid—a MUFA. In metabolic ward studies by Keys et al.[88] and Hegsted et al.,[78] monounsaturates were found to be "neutral" in their action on plasma total cholesterol levels, i.e., monounsaturates neither raised nor lowered the cholesterol levels. This conclusion was based on responses relative to carbohydrates, which likewise were reported to be "neutral" with respect to total cholesterol levels. These general conclusions have been confirmed by Mattson and Grundy.[80] Although the latter workers questioned whether linoleic acid has more LDL-cholesterol lowering action than oleic acid, they obtained results similar to those of previous investigators for oleic acid. In contrast to linoleic acid, high intakes of oleic acid do not reduce concentrations of HDL-cholesterol when it is substituted for SFA.[80-82,114] Overall, therefore, oleic acid appears to have favorable effects on lipoprotein levels, especially when substituted for SFA in the diet.

When carbohydrates replace SFA in the diet, they produce a lowering in total cholesterol[78,88] and LDL-cholesterol.[81,82,114] These reductions in cholesterol levels are essentially equivalent to those of monounsaturates. In short-term studies, triglycerides rise more in response to carbohydrate intake than to intakes of SFA and MUFA. This reflects the "hypertriglyceridemic" response to high-carbohydrate diets reported by Ahrens et al.[115] and Knittle and Ahrens.[116] Limited evidence suggests that the elevation in triglycerides in response to high-carbohydrate diets does not persist for long periods,[117] although this long-term mitigation of increased triglyceride levels has by no means been confirmed. High-carbohydrate diets will reduce HDL-cholesterol concentrations in short-term studies,[81,82,114] and the evidence available suggests that lower HDL levels are a sustained effect in populations consuming high carbohydrate diets.[118] The HDL-lowering effect of high carbohydrate diets extends to apoprotein A-I levels, which also are reduced by high carbohydrate intakes.[102]

In spite of the differences among the various nutrients—MUFA, PUFA, and carbohydrates—on the metabolism of lipoproteins, the differences are not particularly great. Both carbohydrates and polyunsaturates lower LDL-cholesterol levels when substituted for saturates, and at high intakes they lower HDL-cholesterol levels, although the latter response usually is relatively small and may not be of clinical significance. Carbohydrates raise triglyceride concentrations at least in the short term. MUFA may have a favorable effect on all serum lipoproteins, but the most favorable mix of the nutrients (linoleic acid, oleic acid, and carbohydrates) that should be substituted for SFA in the most desirable diet remains to be clarified. It is likely that a considerable range of choices will produce similar effects on plasma lipid and lipoprotein levels.

On the basis of metabolic ward studies alone, the desirable percentage of total fat in the diet cannot be ascertained. In the metabolic ward, a diet moderately high in fat but low in SFA will induce as much lowering of cholesterol levels as one that is low in total fat and SFA and high in carbohydrates. In general, however, lower intakes of total fat will help to reduce the intake of SFA and also facilitate maintenance of desirable body weight. The key to reducing the LDL-cholesterol level is to decrease intakes of SFA; little is gained by reducing unsaturated fatty acid intake to bring total fat intake to very low levels.[80-82,84,119]

A second major cholesterol-raising constituent of the diet is cholesterol itself. Some studies done on outpatients have failed to show that increasing dietary cholesterol raised the level of plasma cholesterol;[120-124] but when investigations are done on the metabolic ward or in tightly supervised settings, where all nutrients can be carefully controlled, increasing dietary cholesterol raises blood total cholesterol and LDL-cholesterol concentrations.[61,78,79,125-130] Responses of individuals to high cholesterol intakes appear to be variable.[131,132] However, this variability is not always reproducible when multiple studies are carried out in individuals.[129,130,133] Therefore the response may be more uniform than revealed by a single study in a group of individuals. Certainly on the average, dietary cholesterol intake raises the blood cholesterol level as concluded in the NIH workshop summarized by Grundy et al.[16] and in the NRC report *Diet and Health: Implications for Reducing Chronic Disease Risk.*[5] The rise occurs mainly in LDL-cholesterol, but VLDL-cholesterol and HDL-cholesterol levels also can increase.[134]

The third cholesterol-raising factor is excess caloric intake resulting in obesity. The effects of obesity on blood cholesterol and lipoprotein levels, however, are complex.[135] Several epidemiologic studies support a positive relationship between body weight and total cholesterol levels; still, this is not a universal finding and confounding variables cannot always be excluded. In a supervised setting, Anderson et al.[136] noted that weight gain generally causes a rise in total cholesterol concentration. Conversely, weight loss in obese individuals has been reported to lower total cholesterol levels.[137-139] Obesity has been associated with increased production of low density lipoprotein.[140] More recently, Davis et al.[141] showed that weight reduction in hypercholesterolemic obese patients caused a significant reduction in LDL-cholesterol levels. Wolf and Grundy[142] were unable to demonstrate that weight loss in obese, normocholesterolemic individuals produced a decrease in LDL-cholesterol concentrations, but they did show a decrease in VLDL-triglyceride and VLDL-cholesterol levels and an increase in HDL-cholesterol concentrations. In this latter study,[142] the diet composition was unchanged before and after weight reduction, which may have minimized any change in LDL-cholesterol concentrations. For example, Caggiula et al.,[143] in the MRFIT, demonstrated a close correlation between changes in body weight and responses in blood cholesterol levels to changes in diet composition. Those individuals who lost weight in addition to reducing intakes of SFA and dietary cholesterol had greater reductions in LDL-cholesterol concentrations than those who merely changed diet composition without weight loss.

The possible relationships between dietary fiber and certain chronic diseases has stimulated much interest in the last decade. Dietary fiber is a generic term to describe substances that are not metabolized by human digestive secretions. Dietary fiber can be categorized as soluble or insoluble. The soluble fibers are pectin and gums. Soluble fibers often retard gastric emptying, possibly because of their gelling properties. Insoluble fibers—e.g., wheat fiber—have a high water-holding capacity and exert laxative effects.

In general, insoluble fibers have no effect on blood cholesterol or lipoprotein levels. Oat bran exhibits hypocholesterolemic properties due to its appreciable content of oat gum.[144] Soluble fibers such as pectin, guar gum, locust bean gum, or psyllium in large quantity supplementation have been shown to lower total and LDL-cholesterol levels.[145-148] The absolute effect on LDL-cholesterol concentrations is modest even when the amount of soluble fiber such as oat bran is consumed in appreciable amounts (60 gm).[149] This effect, however, represents a useful adjunct to an eating pattern low in SFA and cholesterol.

In summary, research in humans has made valuable contributions to the understanding of the effects of diet on the metabolism of cholesterol and lipoproteins, and lends support to many of the public health recommendations for dietary change. In turn, the lipid responses to many recommended dietary changes have been characterized;[150] however, many important diet-lipid questions have not yet been addressed, much less answered.

3. Epidemiologic Research

a. Blood Lipids and CHD

Blood Cholesterol and CHD. Substantial epidemiologic evidence has accumulated over the past 40 years indicating that blood levels of total cholesterol and LDL-cholesterol are highly correlated with severity of coronary atherosclerosis and rates of CHD. Three types of epidemiologic investigations support these relationships and provide strong support for a causal connection. These are (a) within-population studies, (b) cross-cultural studies, and (c) migration studies. Each type is reviewed in the following section.

Within-Population Studies. The most extensive and long-term study of this type has been the Framingham Heart Study. This project began in 1948 and continues to the present time, over 40 years. At the outset, 5,209 men and women living in Framingham, Massachusetts, entered the study. Individuals have been examined every 2 years and have been evaluated for a host of factors that might be related to subsequent development of disease. Over the years, data emerging from this project have demonstrated that age, male sex, elevated blood cholesterol level, high blood pressure, cigarette smoking, obesity, and diabetes mellitus are characteristics that increase the probability for heart attack and stroke.[151] These characteristics, among others, generally have become known as risk factors.[152]

The Framingham study has firmly established high blood cholesterol as a major risk factor for all clinical manifestations of CHD. High blood cholesterol also was found to be a significant predictor of intermittent claudication, although not of stroke or transient cerebral ischemic attacks. When the predictive power of total blood cholesterol is expressed as a risk ratio (the CHD rate at high cholesterol levels divided by the CHD rate at lower cholesterol levels), a high blood cholesterol was a better predictor of CHD in younger men than in older men.[153] However, the number of CHD cases that are attributable to elevated blood cholesterol (attributable risk = the CHD rate at higher cholesterol levels minus the CHD rate at lower cholesterol levels) does not decrease with age.[5] In other words, high blood cholesterol continues to be an important risk factor throughout life. Increased levels of blood cholesterol are very common in the elderly.[154] The Framingham Heart Study has shown that elevated blood cholesterol is also an independent risk factor in women.

In later years and in numerous publications, the Framingham study has extended its analysis of the role not only of total cholesterol but also of cholesterol lipoprotein subfractions and triglycerides.[155,156] LDL-cholesterol levels have been shown to be positively and strongly related to CHD rate, even more so than total cholesterol concentrations. Furthermore, low HDL-cholesterol concentrations are powerful predictors of CHD.[157] More recently, total cholesterol/HDL-cholesterol (and LDL-cholesterol/HDL-cholesterol) ratios were also shown to be useful predictors, indicating important interactions between the lipid risk factors. The predictive value of HDL-cholesterol has been affirmed in many other large studies, including the Lipid Research Clinic's Coronary Primary Prevention Trial (LRC-CPPT) and the MRFIT experience. Although blood triglycerides were positively correlated with CHD rates, earlier data did not reveal an "independent" relationship between triglycerides and CHD.[158] Recently, however, the triglyceride level has emerged as an independent predictor, especially in women, at Framingham.[157] The relationship between blood triglyceride level and CHD risk will be considered in more detail later in this section.

One of the more important findings of the Framingham Heart Study is the fact that blood cholesterol level remained a powerful predictor of CHD over many years of followup.[157] Thirty-year followup data from this study have recently shown not only that incremental cholesterol levels are increasingly predictive of CHD, but they also are correlated with total mortality.[159] For example, participants who had total cholesterol levels below 180 mg/dL (4.65 mmol/L) at entry had the lowest 30-year total mortality. This trend was maintained with progressively higher cholesterol levels. This finding has important implications. Most importantly, a lifelong low level of cholesterol is associated not only with a reduced rate of CHD but also with an increased life expectancy. The issue of possible risks of very low blood cholesterol levels is considered subsequently in this report.

Another major finding of the Framingham study is the multiplicative interaction of risk factors. In other words, the occurrence of several risk factors together produces a greater risk than the sum of individual risks.[151] This means that any given level of blood cholesterol assumes increased significance in the presence of other risk factors. Thus, in the presence of smoking, hypertension, or low HDL-cholesterol, even a near average blood total cholesterol concentration is associated with relatively high risk of CHD. It must be noted that CHD was still the number one killer in the Framingham study in that portion of the population with relatively low cholesterol levels, a fact reflecting, in no small part, the coexistence of other risk factors.

Although the Framingham Heart Study is the best known within-country study carried out in the United States, similar results have been observed with many other studies from this country. The 9-year CHD rates in the Honolulu Heart Program have also shown the same trend related to blood cholesterol level.[160,161] The CHD risk model and data derived from Framingham have been compared with those of the First National Health and Nutrition Examination Survey Epidemiologic Follow-up Study. Using blood cholesterol, systolic blood pressure, and cigarette smoking, the Framingham predictive model was found to be generalizable to this randomized sample of U.S. citizens.[162] The data from several of these surveys were combined in the pooling project.[151] The pooling project data also revealed a strong relationship between total blood cholesterol and CHD; again the correlation was magnified by the presence of other risk factors. However, these studies conducted over a relatively few years did not fully resolve the issue of whether the relationship between cholesterol levels and CHD risk is continuous over a broad range of cholesterol levels from low to high, or whether there is a threshold level, in the range of 200-220 mg/dL (5.17-5.69 mmol/L), only above which does risk for CHD rise appreciably.

This latter issue was partially resolved by the 30-year followup of the Framingham study, which showed that individuals with cholesterol levels below 180 mg/dL (4.65 mmol/L) had lowest rates of CHD and total mortality.[157] Additional evidence on this point has been provided from those men who were screened but not enrolled in the Multiple Risk Factor Intervention Trial (MRFIT). In the followup of this population sample of more than one-third of a million men, ages 35 to 57 years, for whom blood cholesterol measurements were obtained in the early 1970's, there was a strong, positive relationship between total blood cholesterol level and CHD death.[163-165] The increase in CHD mortality was continuous from low cholesterol levels to high, demonstrating that no threshold relation exists between cholesterol levels and CHD risk. This finding has been confirmed in a 10-year followup of the same population[1] (figure 1,

page 7). The relation between cholesterol level and CHD mortality is curvilinear; risk is progressively greater at higher cholesterol levels.

More direct evidence that high blood cholesterol is atherogenic comes from angiographic and autopsy studies in which coronary arteries have been examined. Several investigations have shown that total cholesterol and LDL-cholesterol levels are correlated directly with severity of coronary atherosclerosis as revealed by coronary angiography.[165-168] The same relationship holds for aortic and coronary atherosclerosis in prospective autopsy studies.[169,170] These reports provide strong evidence that high total and LDL-cholesterol levels are directly atherogenic in humans.

The link between blood cholesterol and CHD observed in the United States has been confirmed by epidemiologic studies carried out in other countries.[171-175] The connection is a general relationship, whether occurring in so-called "high-risk" or "low-risk" populations. There appear to be no subpopulations that are immune to the CHD risk-enhancing effect of high cholesterol levels.

In the Israel Ischemic Heart Disease Study, CHD death rates were directly related to the cholesterol level.[176] In Japan, where lower cholesterol levels are common, a clear relationship has also been found with CHD morbidity rates nearly quintupling from the lowest (<180 mg/dL; 4.65 mmol/L) to highest (>220 mg/dL; 5.69 mmol/L) tertiles of cholesterol level.[177] Similar results with steadily increasing CHD mortality rates by cholesterol level quintile have been found in Great Britain.[178] These studies provide further support for the concept that the lower the cholesterol level the greater will be the reduction in CHD risk. Indeed, these studies go even further. They suggest that total blood cholesterol, or more specifically LDL cholesterol, is a fundamental risk factor for development of coronary atherosclerosis. In certain countries such as Japan and China where the average blood cholesterol level has been low, CHD is relatively rare even in the presence of other risk factors. In such countries, a high prevalence of hypertension commonly produces stroke, but does not produce high CHD rates, while a high prevalence of smoking commonly causes lung cancer, but again not a high CHD rate. Even diabetes mellitus or low HDL-cholesterol levels do not greatly increase risk for CHD in populations having very low concentrations of LDL cholesterol. These important epidemiologic observations have led many international groups to recommend national dietary changes as part of population strategies to reduce the prevalence of CHD.[4]

The importance of blood cholesterol as a risk factor for CHD in various populations has been further emphasized by Peto.[179] He has pointed out that typical correlations obtained in epidemiologic studies tend to underestimate the strength of the connection. For example, the Framingham study indicated that for every 1 percent increase in cholesterol level, the risk for CHD increased by 2 percent on the average over the whole range of cholesterol levels. However, according to the calculation proposed by Peto, the duration of the observation plays a role. He has pointed out that analyses of long-term observational studies suggest that a 1 percent increase in cholesterol level may actually be associated with a 3 percent increase in risk. Nonetheless, it must be emphasized that these are *average* relationships, and the impact is less when low cholesterol levels are present and greater with high cholesterol levels.

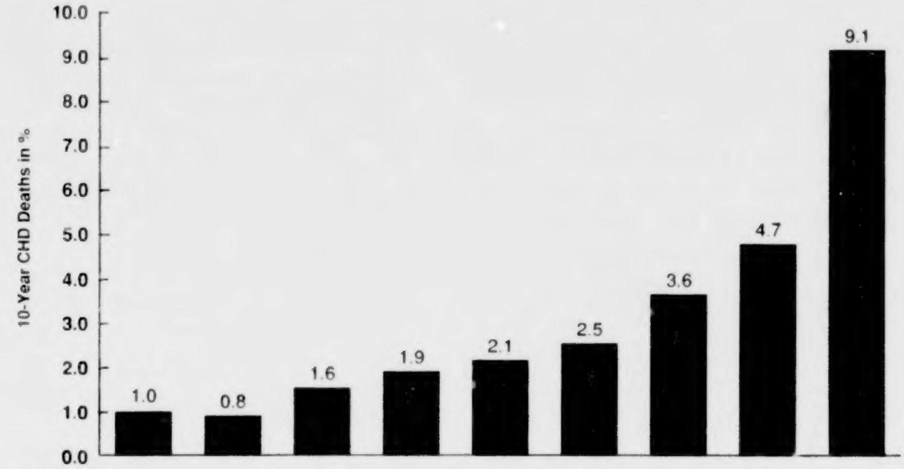
Cross-Population Studies. The most extensive and long-term epidemiologic investigation comparing populations has been the Seven Countries Study.[180] This project was initiated to examine the effects of culture, differences in diet, and other health habits on risk factors (such as cholesterol levels), morbidity, and mortality from CHD. The 16 population groups studied, about 500 to 1,000 persons per cohort, were chosen to represent contrasting experiences with CHD. A total of about 11,000 men, ages 40 to 59 years at entry, were selected from Finland, Greece, Italy, Japan, The Netherlands, the United States, and Yugoslavia. The study was carefully designed and conducted, utilizing standardized procedures for examinations, laboratory testing, quality control, 7-day food records, and chemical analysis of duplicate meals. Blood lipids were determined at the outset and analyzed centrally at the University of Minnesota. The mean total cholesterol levels for the national population samples ranged from 156 to 264 mg/dL (4.03-6.83 mmol/L). The 10-year incidence and mortality from CHD showed that the population age-adjusted risk of death from CHD rose progressively with increasing total cholesterol level of 180 mg/dL (4.65 mmol/L) and above (figure 6). The cholesterol-disease relationship was not statistically significant within the five cohorts that exhibited the lowest level of blood cholesterol (three Italian and two Yugoslav), although longer followup of these cohorts revealed clear, positive relationships between cholesterol and CHD.[181]

The Seven Countries Study provides additional evidence against the concept of a threshold relationship between cholesterol levels and CHD rates, a possibility that was raised by early Framingham and pooling project data. As shown in figure 6, those with cholesterol levels below 200 mg/dL (5.17 mmol/L) had lower CHD rates than those with levels between 220 and 239 mg/dL (5.69-6.18 mmol/L), and the reduction in CHD was even greater when levels were below 180 mg/dL (4.65 mmol/L). These results thus are concordant with those of the 30-year followup of the Framingham study[157] and the 10-year followup of the MRFIT screenees[1] (figure 1, page 7). They all confirm the general dictum: the lower the blood cholesterol level, the lower the CHD risk.

Migration Studies. The classical migration study is the Ni-Hon-San Study.[182] In this study, comparisons were made in populations of Japanese living in Japan, with people originally from the same regions of Japan who were living in Hawaii or in California. The study established rigorous quality

Figure 6

The Seven Countries Study: Total Blood Cholesterol and Percentage of CHD Deaths in Men Originally Free of Cardiovascular Disease



Entry Serum Cholesterol (mg/dL)	< 160	160-179	180-199	200-219	220-239	240-259	260-279	280-299	≥ 300
CHD Events	10	9	25	32	31	29	29	26	60
Men Exposed	1007	1157	1525	1682	1480	1181	806	551	662

Source: Keys, A. *Seven Countries: A Multivariate Analysis of Death and Coronary Heart Disease.* Cambridge, MA: Harvard University Press, 1980.[181]

control of measurement and used standardized CHD prevalence and incidence data. There was a marked difference between all three cohorts in the prevalence of CHD observed at entry—the lowest being in Japan and the highest in California. In a similar pattern the age-adjusted mean total cholesterol was 176 mg/dL (4.55 mmol/L) for the Japanese living in Japan, 219 mg/dL (5.67 mmol/L) for the Hawaiian Japanese, and 226 mg/dL (5.85 mmol/L) for the Japanese in California. These blood cholesterol differences were associated with stepwise increases in CHD. The CHD incidence in California Japanese immigrants was two to three times higher than that of the CHD incidence for the Japanese in Japan.

Similar changes in cholesterol level related to shifts in dietary composition have been noted among groups of people migrating into Israel.[183] Conversely, groups of Irish migrating from Ireland to Boston did not undergo major nutrient intake changes and did not display major differences in blood cholesterol level or in CHD rates.[184]

The migration studies not only support the concept that populations with the lowest cholesterol levels have the lowest rates of CHD, but they also demonstrate that human populations are susceptible to coronary atherosclerosis when blood cholesterol levels are raised by environmental factors. Thus, they add strength to the blood cholesterol-CHD link as revealed by within- and between-population studies.

Blood Triglycerides and CHD. The relation between blood triglycerides and CHD risk is not as well delineated as that between total blood cholesterol (and LDL-cholesterol) and CHD. Even so, most epidemiologic studies show a positive correlation between triglyceride levels and CHD risk, but often only on univariate analysis.[185,186] This relationship may be weakened somewhat because of the lability of triglyceride levels with recent food intake, compared to more constant cholesterol concentrations,[187] and by the likelihood that very high triglyceride levels do not raise risk more than moderately raised concentrations.[188] Moreover, triglyceride levels generally are correlated inversely with HDL-cholesterol levels and directly, but weakly, with total cholesterol concentrations. When the latter two are given priority in multivariate analysis, triglycerides lose much of their ability to predict CHD. Such analyses, however, do not take into consideration the possibility that defects in triglyceride metabolism may be the underlying causes of many cases of elevated cholesterol levels or of reduced HDL-cholesterol. Even if the blood cholesterol serves as a better predictor of CHD, this does not eliminate the possible importance of disorders of triglyceride metabolism in the causation of CHD.[157] Furthermore, there is growing evidence that certain triglyceride-rich lipoproteins, like VLDL remnants, are atherogenic; these lipoproteins are increased in many hypertriglyceridemic states.

Although several epidemiologic studies do not reveal an "independent" correlation between triglyceride concentrations and CHD, as reviewed by Hulley et al.,[185] data from several studies[189,190] as well as recent Framingham data[157] have reported an independent effect of high blood triglyceride levels on CHD risk. This effect may be more pronounced in women. Recently, Avins et al.[186] reviewed over 30 studies that have been published in the 1980's that contain information relevant to this issue. These studies were broadly classified into: cross-sectional studies, prospective cohort studies, and intervention trials. These authors concluded as follows:

> The debate surrounding the independent role of TG [triglycerides] in the pathogenesis of CHD cannot be resolved based on available data. Current evidence indicates that, overall, the association often noted between TG and CHD is frequently due to confounding caused by the correlation between TG and cholesterol subfractions. Unlike the data implicating

increased LDL cholesterol and diminished HDL cholesterol in the causation of coronary artery disease, a strong and consistent thread of epidemiologic evidence does not exist for isolated HTG [hypertriglyceridemia]. However, because of several reports of positive associations even after adjustment for LDL cholesterol and HDL cholesterol, and because inferences based on statistical adjustment are susceptible to statistical modeling and other errors, a definite conclusion cannot be reached at this time.

Nonetheless, even if high blood triglycerides per se are not an independent predictor of CHD risk, available evidence indicates that hypertriglyceridemic patients in general are at increased risk. From a population viewpoint, the most common cause of elevated blood triglyceride levels is obesity. Thus, the tendency of obesity to raise triglyceride and cholesterol levels, and to reduce HDL-cholesterol levels, provides additional reasons to recommend maintenance of a desirable body weight. Another frequent cause of high blood triglycerides is an excessive intake of alcohol, providing an important rationale for people to limit their alcohol consumption.

HDL-Cholesterol and CHD. Most within-country studies show an inverse correlation between HDL-cholesterol concentrations and CHD risk.[157,176,191,192] This inverse relation holds even within countries in which total cholesterol levels and CHD rates are relatively low. In the Framingham study,[193] the relationship of low HDL-cholesterol to CHD for both sexes ages 50 to 80 was found to be inverse and highly significant. The relationship held even into older age groups. Gordon et al.,[194] reviewing four major epidemiologic studies, found that each 1 mg/dL decrement in HDL-cholesterol was associated with an increment in CHD risk of between 2.0 to 3.7 percent in men and 3.0 to 4.7 percent in women.

In spite of these within-country findings, cross-country studies do not reveal as strong a relationship between HDL and CHD as is found between total blood cholesterol (or LDL-cholesterol) and CHD risk.[191] This lesser HDL-CHD connection may have two explanations. First, in populations that have relatively low HDL levels, LDL concentrations likewise tend to be low, and, as indicated before, CHD risk is relatively low when LDL levels are low, even in the presence of other risk factors (including reduced HDL levels). And second, there is not as wide a range of HDL-cholesterol levels between countries, regardless of CHD risk, as there is for LDL-cholesterol levels.

Furthermore, epidemiologic studies have uncovered strong associations between other risk factors for CHD and HDL concentrations. Cigarette smoking, obesity, and lack of exercise are each associated with lower HDL-cholesterol levels. It is difficult to disentangle these confounding variables when assessing the role of HDL in CHD risk. The issue is further complicated by the complex interaction of HDL with other lipoproteins, notably potentially atherogenic, triglyceride-rich lipoproteins (e.g., VLDL remnants) and small, dense LDL.[195] Thus, available data on HDL, taken as a whole, do not provide as strong a rationale for attempting to raise HDL levels in the general population as for lowering LDL levels.[196] They strongly support general recommendations to stop smoking, to eliminate obesity, and to increase exercise—all of these being actions likely to raise HDL-cholesterol concentrations.

b. Diet and CHD

Epidemiologic data provide strong evidence that dietary factors are a major cause of CHD. As reviewed in preceding sections, animal studies first suggested a relationship between diet and atherosclerosis, a relationship mediated through diet-induced hypercholesterolemia. Clinical and metabolic-ward studies have demonstrated that certain dietary factors can raise blood cholesterol levels. Such short-term dietary studies do not, of course, demonstrate the critical link between diet and CHD. Linkage between dietary patterns and coronary atherosclerosis at autopsy has been reported by Moore and colleagues.[197] Ideally, a definitive diet-heart primary prevention trial would prove the diet-CHD connection; but after careful analysis of feasibility, such a trial was judged to be unrealistic for many reasons.[198] While drug trials provide strong evidence that cholesterol lowering will reduce CHD risk, they do not fully answer the diet-heart question.

For many disorders, epidemiologic studies yield clues to possible causal connections between various environmental and ethnic factors and disease endpoints; these possible connections must be substantiated by investigations of other kinds. For the diet-heart issue, the available information from epidemiologic studies has been reinforced by many other kinds of research. The issue no longer belongs in the category of "association," but the totality of data indicate a causal link. The epidemiologic data on the diet-heart connection derives from population samples representing literally millions of people. Thus, by the sheer number of subjects under investigation, it has been possible to minimize the effects of confounding variables.

Epidemiologic studies have three valuable features for assessing the diet-CHD relationship. These are sample size (number), duration, and adherence. Enormous numbers of people can be sampled after a lifetime of adherence to a particular eating pattern.

Estimates of the impact of diet on CHD risk have been made from the three types of epidemiologic studies described previously—within-population, cross-population, and migration studies. During the early part of the 20th century, there were reports that atherosclerotic disease was rare in nonindustrialized countries in contrast with industrialized countries and that the differences varied with the habitual diet of the country. Rosenthal,[199] reviewing this literature up to 1934, deduced a relationship between intake of fat and cholesterol and atherosclerosis. The role of dietary factors in the manifestation of CHD was observed in Norway during World War II when deaths from CHD fell dramatically as the Nazi occupiers diverted dietary fat to munitions production.[200] Although certain within-country studies have provided valuable information, they have a disadvantage in that they contain a relatively narrow range of differences in eating patterns within a single population. On the other hand, wide differences in eating patterns exist between countries and for migrating populations, and thus more meaningful comparisons can be drawn from studies of the latter two types.

As indicated before, three major dietary factors that raise the blood cholesterol level and thus potentially increase risk for CHD have been identified. These are saturated fatty acids, dietary cholesterol, and obesity. Thus, it is appropriate to review epidemiologic data as they pertain to each of these factors and their relation to CHD. In addition, epidemiologic studies provide suggestive evidence about the role of other factors—unsaturated fatty acids, carbohydrates, and fiber—although the available data are not as firm as for the major dietary factors affecting blood lipid levels.

c. Epidemiologic Studies of Diet and CHD

Saturated Fatty Acids. The strongest epidemiologic support that diets high in SFA contribute significantly to increased CHD risk comes from the Seven Countries Study.[180] This study found a high correlation between intakes of SFA and rates of CHD in several different population groups of the seven countries under investigation. The chemical analysis of duplicate diets, collected over four seasons of the year, showed substantial variation in the amount and type of fat among the populations sampled. The saturated fat was low (9 percent of calories or less) in Greece, Italy, Japan, and Yugoslavia, and high (18-20 percent of the calories) in the populations from Finland, The Netherlands, and the United States that were studied. The average cholesterol levels and CHD rates were highly correlated with the percent of calories derived from SFA. In Crete, where the average total fat intake was 40 percent of calories but the SFA intake only 8 percent of calories, there were no CHD deaths and few incident cases after 10 years. This relationship appeared to be mediated through the action of SFA to raise the blood cholesterol level. In the 15-year followup of the Seven Countries Study,[201] intakes of SFA were found to be correlated with total mortality rates as well as with CHD rates.

Other epidemiologic studies have also observed high correlations between dietary SFA and CHD.

One of these was the Ni-Hon-San Study[182,202] described previously. In this rigorous and detailed study, prevalence rates of CHD were found to be lowest in Japan and highest in California. The intake of SFA and cholesterol increased with migration and westernization of the population, and were associated with higher CHD mortality rates.[203,204] Further, CHD rates in the three populations were correlated with blood cholesterol levels. There was a particularly high correlation between percent of calories from dietary SFA and CHD rates in these populations, a finding in accord with the Seven Countries Study.[180] Similar data have been found when other population groups are compared.[205]

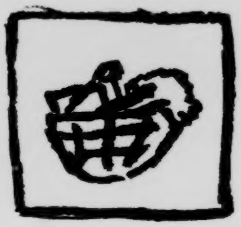
Still other epidemiologic studies showing a correlation between CHD and intake of SFA have been the Western Electric Study,[206,207] the Ireland-Boston Study,[184] the Honolulu Heart Program,[208,209] and the Zutphen Study.[210] Similar observations have been made in migrants to Israel[211] and South Sea Islanders who have moved to industrialized cities in the South Pacific.[212] In each case, changes in dietary habits toward higher SFA and cholesterol consumption were associated with increasing incidence of CHD. Finally, a survey by the Food and Agriculture Organization of the United Nations of CHD rates in 20 countries indicated that intakes of SFA were highly correlated with CHD.[213] Many other smaller epidemiologic studies provide data also supporting a connection between intakes of SFA and rates of CHD.

Populations with specific dietary practices provide additional information. Within the United States, studies of the Seventh Day Adventists show differences in plasma lipid levels and CHD incidence of men according to dietary habit, e.g., nonvegetarian, lacto-ovo-vegetarian, and pure vegetarian. In contrast with a non-Adventist comparison population, nonvegetarian men age 35 years and above had a CHD rate that was 44 percent lower, the lacto-ovo-vegetarian group 57 percent lower, and the pure vegetarian group 77 percent lower.[214] Other studies associate low blood cholesterol levels and low CHD risk among vegetarians.[215-219] Although dietary differences such as low SFA probably account for part of the differences in CHD risk in vegetarians, abstinence from tobacco and alcohol use and other health characteristics are probably also involved.

Dietary Cholesterol. For many years it has been known that feeding cholesterol in the diet to various laboratory animals such as rabbits, chickens, and monkeys produces hypercholesterolemia and atherosclerosis. However, since humans are less susceptible to the blood cholesterol-raising effects of dietary cholesterol than some other animal species, there has been an ongoing debate whether high intakes of cholesterol are an important cause of human atherosclerosis. Indeed, humans can vary in their response to dietary cholesterol.[129,220] Although most epidemiologic studies have stressed the importance of SFA as a dietary atherogenic factor, a growing body of evidence implicates dietary cholesterol as well. This evidence includes results of the Western Electric Study,[206,207] the Honolulu Heart Program,[208] the Ireland-Boston Study,[184] and the Zutphen Study.[210] In all of these studies, intakes of dietary cholesterol were correlated positively with rates of CHD, sometimes independently of blood cholesterol-CHD correlation. In addition, in studies based on the Food and Agriculture Organization and World Health Organization data, mean per capita intakes of dietary cholesterol correlate with CHD mortality rates. It must be noted, however, that intakes of SFA and cholesterol, in general, are highly correlated in populations making it difficult to distinguish clearly the independent effect of each. The epidemiologic data relating dietary cholesterol to human CHD have been reviewed in detail by Stamler and Shekelle.[221] These investigators conclude that "both cross-population and within-population epidemiologic data indicate that dietary cholesterol is atherogenic in humans, over and above—i.e., independent of—its effect on serum cholesterol." This implies that dietary cholesterol may promote atherosclerosis both by raising total blood cholesterol and LDL-cholesterol levels and by mechanisms independent of fasting cholesterol levels, possibly through the formation of cholesterol-rich postprandial lipoproteins (e.g., chylomicron remnants).

Obesity. There is a common belief that obesity contributes to cardiovascular disease. In fact, several reports[222-226] indicate a connection between being overweight and developing CHD. The mechanisms for this connection are not entirely clear, however, because obesity has so many different metabolic effects. It raises blood pressure, increases blood cholesterol, reduces HDL-cholesterol, and induces resistance to the peripheral action of insulin. Any of these factors may heighten CHD risk. Thus, the influence of obesity on CHD risk might be explained entirely by its action to augment known risk factors. On the other hand, data from the Framingham Heart Study[227] indicate that obesity also is an independent risk factor for CHD. This report examined the relationship between the degree of obesity and incidence of cardiovascular disease in more than 5,000 men and women of the original Framingham cohort. These individuals were followed for a period of 26 years. Analyses have shown that the percentage of desirable weight on initial examination, according to Metropolitan relative weight tables, predicted 26-year incidence of coronary disease (both angina pectoris and CHD other than angina) and CHD deaths independent of age, blood cholesterol, blood pressure, cigarette smoking, left ventricular hypertrophy, and glucose intolerance. Moreover, weight gain after the initial measurement also predicted an increased risk for CHD. This important study shows that excess caloric intake leading to obesity is a major dietary factor predisposing to CHD.

Unsaturated Fatty Acids. There are three major categories of unsaturated fatty acids—omega-6 polyunsaturates (mainly linoleic acid), omega-9 monounsaturates (mainly oleic acid), and omega-3 polyunsaturates (linolenic acid, EPA, DHA). In the 1950's and 1960's, there was intense interest in the putative cholesterol-lowering action of linoleic acid, and some investigators of that era advocated high intakes (i.e., 10-20 percent of total calories). However, no large populations have consumed such high intakes of linoleic acid for prolonged periods, and, thus, the long-term consequences of such intakes are not known. Accordingly, there are no epidemiologic results to support very high intakes of linoleic acid. Consequently, most expert panels have recommended that individuals consume no more than 10 percent of calories as polyunsaturated fatty acids. That this is prudent is bolstered by clinical trial data suggesting that high intakes of linoleic acid may increase risk for gallstones[228] and animal data that high PUFA intake may promote development of tumors[229,230] and suppress the immune system.[231]



On the other hand, very low intakes of linoleic acid (less than 5 percent of calories) have been related to increased CHD rates in high-risk populations.[232-235] The Western Electric Study[206] also noted that modest increases in linoleic-acid intake were associated with decreased CHD rates. All of these data provide some justification for the recommendation[5] that omega-6 PUFA intake be maintained at about the current level (approximately 7 percent of calories). There are no epidemiologic data to suggest that intakes in the range of 5-10 percent of calories are accompanied by an increased risk of diseases such as cancer or gallstones.

Substitution of dietary saturated fatty acids with oleic acid results in a fall in LDL-cholesterol levels. However, in contrast to linoleic acid, there are epidemiologic data that demonstrate long-term safety of high intakes of oleic acid. In the Mediterranean region of the world, where large quantities of olive oil are consumed, rates of CHD are relatively low, and there is no evidence for harmful effects (e.g., increased rates of cancer). In fact, in the 15-year followup of the Seven Countries Study,[201] intakes of monounsaturated fatty acids (oleic acid) were inversely correlated with both CHD rate and total mortality. This finding thus justifies encouraging higher intakes of oleic acid in the diet, i.e., 14-16 percent of calories, as a substitute for saturated fatty acids.

In recent years, there has been a growing interest in the possible beneficial effects of omega-3 polyunsaturated fatty acids. This interest stems, in part, from the limited observations suggesting that Greenland Eskimos, who consume large quantities of omega-3 PUFA, have low rates of CHD.[236] These fatty acids are known to have an antithrombotic effect; many Greenland Eskimos have a bleeding tendency. The low risk of CHD in this population thus might be explained by the absence of coronary thrombosis or perhaps some other protective effect of omega-3 PUFA. On the other hand, there could be a variety of confounding factors in the Eskimo population that could contribute to low rates of CHD. For example, they generally have low levels of other major risk factors. Nonetheless, some other population studies[237,238] suggest that consumption of fish may exert a protective action against CHD. Although these population studies alone do not show a clear protective effect of omega-3 PUFA, they provide another reason for a more liberal intake of fish, especially because fish contain protein with low proportions of saturated fatty acids.

Carbohydrates. There are no large population studies which suggest that high intakes of carbohydrates are accompanied by increased risk for CHD. If anything, the opposite is true. Populations that consume high-carbohydrate, low-fat diets generally have low rates of CHD.[180] This finding is in accord with metabolic studies which show that replacement of saturated fatty acids by carbohydrates causes a lowering of total cholesterol and LDL-cholesterol levels. As reviewed in a previous section, metabolic studies indicate that high-carbohydrate diets can raise triglyceride levels and lower HDL-cholesterol levels,[239] but these changes apparently do not translate into increased risk for CHD.[240] Thus, cross-country, epidemiologic studies in general support the conclusion that intakes of carbohydrates in the range of 55 percent of total calories are acceptable and that low-fat, high-carbohydrate diets are associated with low rates of CHD and of certain forms of cancer.[5,241]

In summary, a broad base of epidemiologic data is available to support the conclusion that there are strong and causal relationships between diet, blood cholesterol level, and CHD. These data provide overwhelming evidence that elevated levels of blood cholesterol and LDL-cholesterol are major risk factors for CHD. In addition, they indicate that dietary composition is highly correlated with rates of CHD. In particular they support the conclusion that three major dietary influences on blood cholesterol level—saturated fatty acids, cholesterol, and maintenance of desirable body weight—directly affect CHD rates.

4. Observations in Special Populations

a. Children

Children represent an extremely important special population. A separate Expert Panel on Blood Cholesterol Levels in Children and Adolescents will provide more detail about how to implement eating pattern changes in healthy children and will recommend approaches for detection and treatment of children with high-risk cholesterol levels. In its report, that panel will discuss more fully the issues of dietary safety and adequacy for children and adolescents. The present report reviews the evidence in summary fashion.

Surveys of childhood levels of cholesterol and lipoprotein fractions show that North American children have higher total and LDL-cholesterol levels than children in countries where atherosclerosis is less frequent.[242] A number of surveys of childhood lipid and lipoprotein levels have been published in the United States. One survey that provides nearly a complete age span and values for the lipoprotein fractions is that of the Lipid Research Clinics (LRC) Prevalence Study.[243] The LRC study sampled populations from multiple sites in the United States.

The blood cholesterol levels in children and adolescents are significantly lower than those in adults. In pubertal years, there is a slight transient fall in total cholesterol and LDL-cholesterol levels presumably due to hormonal changes. Blood cholesterol levels in children have a predictive value for levels of cholesterol in young adult life.

In general, children with high cholesterol levels are most likely to have high cholesterol levels as young adults.[244-246] However, there is considerable variability. Of children whose blood cholesterol levels are greater than the 90th percentile during childhood, most of them will remain above the 50th percentile.[246] A correlation between children's habitual dietary intake of fat and blood cholesterol levels has been shown.[247]

Since no groups of children have been followed for decades after having had their cholesterol or lipoprotein fractions measured, the relationship of high total cholesterol, LDL-cholesterol, and low HDL-cholesterol during childhood and the atherosclerotic process must be inferred from other data.

Obesity plays an important role in the development of hyperlipidemia, hypertension, diabetes mellitus, and other chronic illnesses in adulthood.[248,249] Obesity acquired in childhood most often persists into adult life.[246,250] Obese children generally have higher blood cholesterol levels.[251] In studies following children from school age to age 25 to 30 years, those who gained excess weight for their heights were found to have the greatest increases in their cholesterol levels.[246]

Several lines of evidence point to a genetic role in the hypercholesterolemia of some children. Children with homozygous familial hypercholesterolemia with extreme elevation of total cholesterol and LDL-cholesterol levels have been shown to develop CHD at a very early age.[252] In addition, many children whose cholesterol levels were greater than the population-based 95th percentile had a familial prevalence of hyperlipidemia as well as premature CHD.[253,254] When the progeny of young ischemic heart disease victims were examined, many had hypercholesterolemia.[255] Morrison et al.[256] found that children whose parents had experienced a myocardial infarction earlier than age 50 had significantly higher blood cholesterol levels, on average about 195 versus 175 mg/dL (5.04 versus 4.53 mmol/L), than children with parents without early myocardial infarction.

Fatty streaks are found in the vascular system of most children, regardless of age, race, sex, or environment, by the age of 10 years.[257] These fatty streaks cause little or no obstruction and therefore no clinical symptoms. Since these lesions are ubiquitous, some have questioned whether these are the earliest manifestations of the atherosclerotic process. Newman and colleagues[170] conducted a postmortem study of children whose lipids and lipoproteins had been previously measured and who died accidentally. They showed a relationship between the amount of fatty deposits in arteries and LDL-cholesterol levels. The children with larger proportions of aortic wall involved with fatty streaks were those with higher total cholesterol and LDL-cholesterol levels. Intensive postmortem examination of large numbers of other children has shown that a progression of lesions could be traced from the fatty streaks in coronary arteries to the fibrous plaques.[258] These observations suggest that many coronary artery fatty streaks and some aortic fatty streaks in children are converted to fibrous plaques in adolescents and young adults. Fibrous plaques are the lesions most characteristic of progressing atherosclerosis.[259] With age, the fibrous plaques may become vascularized, calcified, and more obstructive. Autopsy studies of U.S. war casualties in Korea and Vietnam confirmed the frequent presence of grossly detectable coronary artery lesions in late teenage and early adulthood.[260,261] This was also confirmed in similar-age young people dying of accidental death in the United States.[257]

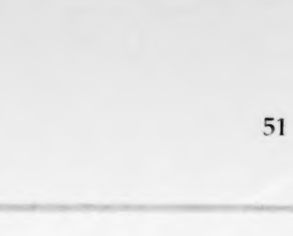
Since the early stages of the atherosclerotic process often have origins in childhood,[262] dietary changes must be viewed in the context of the important nutrient needs for the growth and development of a child. The dietary requirements for a growing child must provide more calories per kilogram of body weight for heat production to offset the relatively high heat losses over a larger surface area per kilogram for children compared to adults.[263,264] In addition, bodily growth in children requires not only further augmentation of caloric intake, but also intake of a diversity of foods that provide the nutrients needed for growth. Particular attention has been drawn to calcium, iron, and zinc.[265] Shifts away from fats, which provide a high caloric density, require special care to assure adequate intake of recommended daily nutrients. Reliance on foods that are low in fat and calories requires that children, in order to eat a nutritionally adequate diet, spend the necessary time to consume more food.

An important question concerns the age at which fat consumption can safely be reduced. In infants whose total nutrition is dependent on milk, there is evidence that very low-fat or skim milk formulas do not provide sufficient energy for normal growth.[266] However, infants fed 30 percent or more of their total calories as fat grow normally.[267] In countries of the Mediterranean and Far East, children eat with apparent safety a diet containing an average of 10 percent or less of calories from SFA and characterized by consumption of grains, legumes, vegetables, fruits, fish, lean meats, and poultry. The human is capable of synthesizing most of the lipid compounds necessary for health. The fatty acids essential for normal growth and development, which cannot be synthesized de novo by humans, are the omega-6 PUFA (e.g., linoleic) and the omega-3 PUFA. Furthermore, lipid-soluble vitamins require fat intake for their absorption.

Surveys of childhood diets were carried out by the National Center for Health Statistics from 1971 to 1974 and again from 1976 to 1980.[268,269] These surveys showed that mean fat intake was about 32-33 percent of total calories consumed per day in the age group 6 to 12 months, and was about 35-37 percent of calories from 1 to 18 years, with approximately one-third of children from 1 to 18 years eating more than 40 percent of calories from fat. Within this same age span, 79 percent received ≥10 percent of their calories from saturated fats. The data from the NHANES II[270] survey showed that 30 percent of children 1.1 to 1.9 years of age consumed more than 300 mg dietary cholesterol per day. For those 2 to 18 years old the mean cholesterol intake was about 280 mg/day, with 34 percent having an intake exceeding 300 mg/day.[271] Thus, many U.S. children, along with many adults, presently consume diets that are rich in SFA and cholesterol.[268,269]

The data related to childhood have been interpreted by expert panels with different emphases. The first expert group to recommend that infants and children modify their diets to prevent CHD was the Inter-Society Commission for Heart Disease Resources.[8] The American Academy of Pediatrics (AAP) has recommended an individual approach. The AAP committee on nutrition statement, "Prudent Life-style for Children: Dietary Fat and Cholesterol,"[272] has concentrated on efforts to identify children with a family history of heart disease or high blood lipid levels in a family member younger than 60 years of age. In addition, the AAP suggests that children with blood cholesterol >176 mg/dL (4.55 mmol/L) be given dietary intervention.[273] This strategy requires individual nutritional, therapeutic, and genetic counseling programs.

In 1983 the American Heart Association adopted the public health approach to preventing CHD in children. The AHA statement, "Diet in the Healthy Child,"[274] recommended that all children over 2 years reduce fat intake to approximately 30 percent of calories with 10 percent or less from SFA, less than 10 percent from PUFA, about 10 percent as MUFA, and cholesterol intake to 100 mg/1,000 calories. This recommendation by the AHA was endorsed by a consensus development panel on lowering blood cholesterol to prevent heart disease that was convened by the National Institutes of Health.[275] These reports view the high incidence of CHD in the U.S. population and the high cholesterol distribution in American children as justification for this dietary intervention. In addition, they suggest that dietary changes in early life and in the family setting have the potential for healthier lifestyles and decreased CHD risk in adulthood.



The positions of the committees on nutrition of the AHA and the AAP have many more similarities than differences. Some critics of the AHA statement have suggested that there is no evidence that the recommended diet is adequate to support growth and development. Some have noted that the diet recommended is bulky and low in energy and essential nutrients because it restricts fat and SFA and promotes an increase in consumption of cereal grains and plant products. Concern has been expressed about the adequacy of intake of high-quality protein, iron, calcium, and other minerals in such diets.[276] However, there is no evidence in the scientific literature to support a conclusion that the decreased intake of fat, SFA, and cholesterol to the levels suggested by the AHA would compromise growth or neurological development. In fact, the diet recommended by the AHA maintains protein and essential mineral and vitamin intake with increased carbohydrate consumption substituting for fat and SFA intake. Recently the committees on nutrition of the AHA and the AAP[277] met and agreed on several points summarized in the following:

1. No changes in the current feeding pattern for infants and children less than 2 years of age are needed.

2. The present trend toward lower SFA intake in diets for children 2 to 18 years of age should be encouraged. The AHA and AAP committees stated that, although it is not unanimous, there is consensus that 30 percent of total calories from fat (with 10 percent from SFA) is a reasonable target for children.

3. Educational programs for physicians and the public should be undertaken so as to discourage excessive limitation of fat and animal protein intake that might lead to mineral deficiencies and growth failure.

4. Research is under way and should be continued on the specific effects on growth and development of diets containing approximately 30 percent of calories from fat.

Many American families have already changed their eating habits toward lower-fat eating patterns without evident untoward effects. Only in children with severe fat-malabsorption syndromes have linoleic acid or fat-soluble vitamin deficiencies occurred.[278] There have been reports of a few zealous families that over-restricted fat and protein intake and whose very young children under 2 years of age consequently failed to thrive.[279] Thus, as dietary changes are recommended to the general population, some caution must be observed with respect to adequacy of energy intake for children. However, a modest reduction of total fat in the diet to 30 percent of calories will allow sufficient energy for growth and development.

The *Diet and Health* report of the NRC,[5] based on its extensive review of the relevant scientific evidence, has recommended that children over the age of 2 years follow the same dietary guidelines as adults. Specifically, the NRC recommends that both children over the age of 2 years and adults consume 30 percent or less of calories as fat, with less than 10 percent of calories from SFA. The NRC has also recommended that children limit their dietary cholesterol intake to under 100 mg per 1,000 calories consumed, with the total not to exceed 300 mg per day.

In summary, data derived from children and adolescents indicate that diet influences blood cholesterol level and that elevated blood cholesterol is often associated with early stages of the atherosclerotic process. Accordingly, it appears reasonable for children, ages 2 years and older, to have SFA, total fat, and cholesterol intakes at the same levels recommended for the adult population. Available data support the safety of following these recommendations. A number of professional and scientific organizations have already made these recommendations.

b. Older Adults

Elderly people form an increasing proportion of the U.S. population. In this age group, CHD is a leading cause of death and disability. Increased levels of blood cholesterol are also common in the elderly,[280] and are frequently higher than the levels at which treatment is recommended by the Adult

Treatment Panel of the NCEP. Thus, there is a need for information and recommendations concerning blood cholesterol in the population of older adults.

Little is also known about older adults' awareness of blood cholesterol levels and CHD, or of the cholesterol levels above which medical treatment is commonly initiated in this population. Population surveys in urban Minnesota have shown that 12.4 percent of men and 13.9 percent of women ages 60 to 74 years were aware of their own diagnosis of hypercholesterolemia;[281] relatively few were under any medical treatment.

The absolute rates of CHD are also high among the elderly population. CHD mortality rates in the 65 and above age categories are many times those found among middle-age adults.[282] In addition, CHD rates, which are much higher in men than women in younger age categories, tend to equalize in older age groups, with women exhibiting considerable rate increases as they age. A substantial burden of CHD morbidity and mortality in the elderly is among women. Because of the relatively high event rate among older people, small percentage shifts in disease rates may produce large differences in number of cases. For example, even a 1 percent fall in CHD mortality rates in the 65 and over age category could result in 4,300 fewer CHD deaths a year in the United States.

Mean blood cholesterol levels tend to rise with age among young and middle-age adults in the United States. For men, they peak in the sixth decade and then decline. For women, the average blood cholesterol level continues to rise into the seventh decade and then decreases slightly.[243,283]

Increased blood cholesterol, by the standards of the ATP, is very common among the older population.[283] Based on the two criteria of the ATP of $\geq$200 mg/dL ($\geq$5.17 mmol/L) and $\geq$240 mg/dL ($\geq$6.21 mmol/L), substantial numbers of individuals will be classified as having borderline-high or high blood cholesterol levels. For men and women, this would include the majority of the population age 60 to 89 years.

Epidemiologic data for persons over the age of 65 years are sparse. Data from early studies indicated little association between total blood cholesterol and CHD mortality in groups over 60 years of age. Long-term followup (12 years) of a population of white males in Los Angeles studied from 1950 to 1962 suggested that blood cholesterol level was unrelated to myocardial infarction rates in older men. Compared with men with a blood cholesterol less than 270 mg/dL (6.98 mmol/L), the relative risk in men above 270 mg/dL (6.98 mmol/L) from ages 21 to 59 years ranged from 1.6 to 3.9. However, in the age category of 60 to 70 years, there was no increased risk (relative risk = 1.0) associated with a cholesterol level over 270 mg/dL (6.98 mmol/L).[284]

Gofman et al.[285] reported that blood total cholesterol lost its predictive power as men aged and that it disappeared as a risk factor in men above age 65. A Danish cohort studied in the same era demonstrated some predictive power for cholesterol, even in the 70 to 80 year age group.[286] The effect, however, was much weaker than that observed for younger men.

Studies conducted more recently, however, including further followup data on previously examined populations, indicate that both total cholesterol and the lipoprotein cholesterol subfractions are predictive of disease in the elderly. In contrast to earlier reports from the Framingham study, which showed little predictive power for cholesterol in the elderly, the 30-year followup data from this study indicate that blood cholesterol levels continue to predict CHD incidence and mortality in older men and women but that the strength of the association lessens with age. Both elevated LDL-cholesterol and reduced HDL-cholesterol were predictive of CHD in men and women through age 82.[287]

In the Puerto Rico heart study, elevated cholesterol $\geq$200 mg/dL ($\geq$5.17 mmol/L) at baseline was found to be predictive of CHD mortality in the male 60- to 79-year age group. The augmentation in risk was only slightly lower than that found in the age group less than 60 years.[288] The Lipid Research Clinics (LRC) prevalence study surveyed healthy populations from 1972 to 1974. Followup data from one center showed that cholesterol predicted ischemic heart disease mortality in the 50 to

64 age group and in the older 65 to 79 group.[289] More recent data from the LRC program for men 65 years or older followed for an average of 8.5 years found that both total cholesterol and LDL-cholesterol were predictive of CHD outcomes after adjustment for other risk characteristics.[290] Similarly, Benfante and Reed[291] have analyzed blood cholesterol and CHD in males over 65 years of age who were followed for an average of 12 years. The incidence of CHD increased steadily from the lowest to highest quartiles of cholesterol level.

Gordon and Rifkind[292] examined the issue of age and cholesterol as CHD risk factors in the data from the 356,222 males, ages 35 to 57 years old, screened for the Multiple Risk Factor Intervention Trial. This analysis shows that CHD mortality rates increased progressively with serum cholesterol within each age group. It also documents that the relationship is greatest in the youngest age group and declines with increasing age. Gordon and Rifkind state that although a smaller proportion of CHD cases may be prevented by cholesterol reduction in older than in younger patients, the potential benefit of treatment, when expressed as the number of events prevented per year per 1,000 treated, is greater in older than in younger patients. Between ages 65 and 74 years, 16 percent fewer men (38 versus 54 percent) and 9 percent fewer women (21 versus 30 percent) would be expected to have a new manifestation of CHD with a cholesterol level of 200 mg/dL (5.17 mmol/L) than with a cholesterol of 285 mg/dL (7.37 mmol/L). A report by Garber et al.[293] suggests that the magnitude of benefit of blood cholesterol lowering in the elderly remains to be fully clarified.

The question of modifying cholesterol levels in older adults is a difficult one with fewer data available than for younger adults. Among the few dietary intervention studies aimed at lowering blood cholesterol levels and CHD, some have used study populations of elderly subjects. Albanese et al.[294] studied nursing home populations age 70 and older to test the effect of a polyunsaturated to saturated fatty acid ratio (P/S ratio) of 0.7, compared with a P/S ratio of 0.1 to 0.3, which was estimated to be the norm for institutional diets at that time. The dietary changes resulted in a fall in blood cholesterol that averaged 20 percent. In the same era, an 8-year controlled dietary trial in which an institutionalized population of men substituted vegetable for animal fats was begun in a Los Angeles Veterans Administration hospital. The study suffered from poor adherence rates and small sample size. However, blood cholesterol was reduced, and there was a reduction in disease rates in the under-65 population. In the older group (>65 years) there were only small differences between the experimental and control groups.[295]

The issue of adequate nutrition in the elderly is important. Both poor dietary intake and malabsorption of important nutrients have been found in many elderly people.[296] Accordingly, there is a concern that some older people may over-adhere to an eating plan that is lower in total fat, SFA, and cholesterol and hence suffer from lack of essential nutrients. However, in a recent series of papers concerning the nutritional status of non-institutionalized elderly people (ages 60 to 93 years), this did not occur (e.g., Goodwin et al.).[297] In this group, individuals who were concerned about lowering blood cholesterol levels and who followed dietary principles to achieve this were not deficient in intakes of protein, calcium, and fat-soluble vitamins. Thus it appears that for this group, the diet both lowered consumption of fats and preserved the intake of essential nutrients.

In summary, population data show that both total blood cholesterol and LDL-cholesterol levels are predictive of CHD events among adults older than 60 years, that CHD is highly prevalent in the elderly, that blood cholesterol levels are elevated in the majority of this group, and that blood cholesterol levels can be reduced by dietary change.

c. Women

Women's dietary intakes and overall health with respect to CHD have only recently received the attention they deserve. CHD accounts for about 250,000 deaths each year among women in the United States, and is responsible for about one-third of all deaths of women in the United States. In age-adjusted data, CHD is the leading cause of death among women, and it is the second most common cause of death among women 35 to 74 years of age. Rates of death due to CHD are 1.5 times higher among black women than among white women.

The trend in the CHD death rate in the 1940's and 1950's was upward among men and older women, flat among middle-age women, and downward among younger women. The decline between 1962 and 1978 in CHD death rates began earlier among women than among men, and the percentage decline in CHD mortality has been steeper for younger than for older women.[298] However, since 1979, declines in CHD mortality have been steeper among men than women, lowering the male/female ratio of CHD deaths for the first time.

First coronary events are more often fatal among women (39 percent) than among men (31 percent).[299] Other initial manifestations of CHD are dissimilar in men and women. Fifty percent of men with CHD are first seen with myocardial infarction, compared with 34 percent of women.[299] In the Framingham study, angina was the initial manifestation of CHD in women more frequently than in men. Although women experience an age-adjusted CHD mortality rate less than one-half the rate among men, the total CHD rate in women is similar to that in men. Women in 1980 were responsible for 58 percent of total expenditures for heart disease.

The risk factors for CHD in women are the same as in men, namely, age, hypertension, cigarette smoking, high total or LDL-cholesterol level, low HDL-cholesterol level, diabetes mellitus, and obesity. However, diabetes appears to be relatively more important for women. Cigarette smoking is a major risk factor, but somewhat less so than in men.[300] Additional characteristics unique to women that may exacerbate the effect of selected risk factors include oral contraceptive use, menopause, hysterectomy, and exogenous postmenopausal hormone use. According to the second National Health and Nutrition Examination Survey (NHANES II), more than 58 percent of U.S. adult women have blood cholesterol levels that are above 200 mg/dL (5.17 mmol/L).[2] Other cardiac risk factors are also common, including hypertension and smoking.[300]

Considerable data on risk factors and CHD associations in women are available from the Framingham study. For women ages 40 to 49 years, smoking and relative weight were predictors of CHD. Systolic blood pressure and blood cholesterol concentration both were positively related but did not reach statistical significance, probably because of the small number of CHD cases. Among women ages 50 to 59 years at baseline, systolic blood pressure, relative weight, and blood cholesterol levels were significant predictors of total CHD.[301] For women age 60 to 69 years, glucose intolerance and high blood pressure (systolic blood pressure) were independently related to the development of CHD.[302]

It has been argued that at least a portion of the sex difference in CHD susceptibility is related to sex differences in blood lipoprotein levels. In men, fasting levels of VLDL-cholesterol and triglycerides rise to a peak at about 45 to 50 years of age and then decline. In women, VLDL-cholesterol and triglycerides rise steadily with age and are lower than in men at all ages up to about 60 years. LDL-cholesterol rises with age in men, but the increase peaks in the sixth decade. In young women, LDL-cholesterol is lower than in men, but rises rapidly with age, overtaking levels in men in the sixth decade. In adults, HDL-cholesterol shows little variation with age, being consistently lower in men. The sex difference in HDL remains even after differences in body weight, smoking, and physical activity have been taken into account.[303]

The data are inconclusive regarding the effect of exercise on plasma cholesterol and lipoprotein levels in women. A few studies report a significant increase in HDL-cholesterol levels in women undergoing strenuous endurance training programs.[304,305] Other studies have reported no effect.[306] It is hypothesized that HDL-cholesterol levels may not increase in women who exercise because of the decreased circulating estrogen concentrations that are associated with endurance training of women.

Although CHD accounts for one-third of all deaths among American women, evaluations of medical therapies among women with CHD have been limited. Clinical trial data to establish the benefit of reducing blood cholesterol levels in women are not available. Expectations about the effects of intervention to prevent CHD among women will need to be based for the most part on the result of available data from trials conducted among men, as well as on inferences from the extensive observational epidemiologic research concerning women.

The data on the prevalence of high blood cholesterol and CHD in women, and data on the nutrient needs of women, particularly adolescents and women of childbearing age, emphasize the importance of dietary recommendations to lower high blood cholesterol levels in women. The data also indicate that needed dietary changes can be accomplished while ensuring full nutritional adequacy.

d. Socioeconomic Status Groups

Socioeconomic status (SES) is usually defined by the level of one or more of the following variables: income, education, and occupation. The levels of these three variables are highly correlated and interrelated. A substantial minority of the U.S. population are members of low SES groups. For example, approximately 15 percent live in poverty defined by family income levels.[307] Poverty rates vary significantly by race, however: 11 percent of whites live in poverty, compared to 34 percent of blacks and 30 percent of Hispanics.

Numerous studies have examined the relationship between SES and risk of developing CHD.[308-315] Some studies have found higher CHD rates among higher SES groups while others have found little relationship with SES, and still others have found an inverse relationship. Some studies have shown different relationships between SES and risk of developing CHD for men and women. Others have found different relationships among different racial groups. Some studies have shown a reversal over time in terms of which SES groups are at higher risk of CHD. In general, lower SES groups currently appear to have higher CHD mortality rates than do higher SES groups. This increase in relative risk is generally in the range of 1.25 to 1.50. Mortality rates for CHD have fallen among all SES groups in the United States in the past 20 years, but the declines have been greater in the higher SES groups.

Part of the differences in CHD mortality rates among different SES groups may be explained by differences in the prevalence of some risk factors and by changes in that prevalence over time.[316,317] Higher prevalence rates of uncontrolled high blood pressure, smoking, physical inactivity, and obesity are currently found among lower socioeconomic status groups.[318,319] In contrast to other risk factors, mean blood cholesterol levels are quite similar in both higher and lower SES groups in the United States.[320] The 1976-80 NHANES II found that mean blood cholesterol levels were about 6 percent lower in both white and black men who live in poverty compared to the levels in white and black men with family incomes above $20,000. Because of the apparent increase in other risk factors and CHD in lower SES groups, reduction in blood cholesterol may be particularly important in this population.

5. Intervention Studies

a. Clinical Trials

The broad spectrum of data reviewed in this report suggests that elevated levels of cholesterol, whatever their cause, lead to accelerated atherosclerosis and CHD, and that a high intake of saturated fatty acids and cholesterol leads to high blood cholesterol levels. The data strongly suggest that lowering blood cholesterol would reduce the incidence of CHD. Until recently though, direct evidence from well-conducted clinical trials that lowering cholesterol levels is safe and beneficial was not available.

Given the role that diet is held to have in preventing CHD, the ideal study of cholesterol lowering would be a clinical trial of diet. This possibility was considered by the Arteriosclerosis Task Force of the National Heart and Lung Institute in 1971. The task force had available to them the results and conclusions of the Diet/Heart Feasibility Study. The Arteriosclerosis Task Force recommended against a dietary clinical trial based on several considerations. For example, the required sample size was variously estimated to range from 24,000 to 115,000 individuals, and 7 to 10 years of followup would be necessary. The managerial problems of carrying out a well-controlled study under such circumstances would make the study difficult to complete. The projections of costs then ranged up to $1 billion. Of special importance was the possibility that subjects adhering to a dietary regime would most likely modify other risk factors, thus rendering interpretation of a positive outcome extremely difficult. It should be noted that these considerations equally apply now, just as they did in 1971, and that it is most unlikely that such a trial will ever be performed. Accordingly, testing the hypothesis that cholesterol lowering will reduce the risk of CHD has largely been attempted through drug studies in relatively high-risk segments of the population.

There are several ways by which blood cholesterol levels can be reduced pharmacologically.[321] A number of effective cholesterol-lowering agents, many acting by different mechanisms, are now available for clinical use. The use and mechanisms of action of these agents have been discussed in the NCEP Adult Treatment Panel Report.[3]

Prior to the 1980's there were many clinical trials of cholesterol lowering by diet or drugs in patients with or without preexisting clinical CHD. Although the results of some of these studies were encouraging, none of them was held to be conclusive. The limitations of these studies included small sample size, a short period of followup, inadequate randomization procedures, absence of a double-blind design, high dropout rates, or problems in statistical analysis. However, a number of well-designed major studies have been reported more recently.

One of these, the Lipid Research Clinics Coronary Primary Prevention Trial, was directed by the National Heart, Lung, and Blood Institute. The LRC-CPPT tested the efficacy of cholesterol lowering in reducing the risk of CHD in 3,806 asymptomatic middle-age men with hypercholesterolemia.[322,323] The treatment group received the bile acid sequestrant cholestyramine resin, and the control group received a placebo for an average of 7.4 years. Both groups followed a moderate cholesterol-lowering diet. The cholestyramine group had 8.5 percent and 12.6 percent greater reductions, respectively, in total and LDL-cholesterol levels compared with the placebo group. The cholestyramine group experienced a 19 percent reduction in risk ($p<0.05$) of the primary endpoint—definite CHD death and/or definite nonfatal myocardial infarction. Further analysis within the cholestyramine-treated group in the LRC-CPPT showed that men who took the full dose of the medication had a fall of 25 percent in total cholesterol (with an average 35 percent decrease in LDL-cholesterol) and had a CHD incidence that was half that of men who remained at pretreatment levels. In addition, the incidence rates for new positive exercise tests, angina, and coronary bypass surgery were reduced by 25, 20, and 21 percent, respectively, in the cholestyramine group. Taking all endpoints into account, 577 of the 1,740 subjects taking placebo developed an endpoint, compared with 477 of 1,741 men taking cholestyramine.[324] It was concluded from these findings that reducing total cholesterol by lowering LDL-cholesterol levels can diminish the incidence of CHD morbidity and mortality in men at high risk for CHD because of elevated total and LDL-cholesterol levels.

Another primary prevention trial using a lipid-altering drug has recently been reported.[325,326] The Helsinki Heart Study was also a randomized, double-blind, placebo-controlled study involving

4,081 middle-aged men in a 5-year followup. To qualify, participants had to have concentrations of LDL-cholesterol plus VLDL-cholesterol greater than 200 mg/dL (5.17 mmol/L). The study assessed the efficacy of treatment with 600 mg of gemfibrozil twice daily in reducing CHD incidence. Treatment resulted in, on average, 10 percent reductions in total and LDL-cholesterol, a 35 percent decrease in triglycerides, and a 10 percent increase in HDL-cholesterol. The cumulative combined incidence of fatal and nonfatal myocardial infarction and cardiac death, the principal endpoint, was reduced by 34 percent in the gemfibrozil-treated group ($p<0.02$). Further analysis within the drug-treated group showed that while decreases in LDL-cholesterol levels were associated with a decreased risk of CHD, the subset in whom LDL-cholesterol did not decrease may also have benefited from the lower CHD rate, possibly because of the associated HDL-cholesterol increase.

The studies described above show the benefits of cholesterol lowering on clinical events. Their findings have been complemented by several recent studies that have focused attention on the impact of cholesterol-lowering on the coronary atherosclerotic lesion itself as seen through angiographic studies. Uncontrolled or partially-controlled angiographic studies using either diet or drugs to modify lipid concentrations had reported decreased progression of coronary lesions. These findings have been confirmed in two controlled trials, the NHLBI Type II Coronary Intervention Study and the Cholesterol-Lowering Atherosclerosis Study.[327,328] In the latter, marked lipid changes over 2 years produced by a combination of a resin (colestipol hydrochloride) with niacin therapy and a strict diet resulted in less progression, greater stabilization, and apparent regression of some coronary lesions. These benefits were observed both in aortocoronary bypass grafts and in native coronary vessels, and were irrespective of pretreatment cholesterol levels. More recent work by Brown et al.[329] lends additional support that atherosclerosis progression can be slowed and regression induced by aggressive cholesterol-lowering therapy in men with coronary artery disease.

Two major studies have reported on the effect of the simultaneous intervention on several CHD risk factors including diet-induced cholesterol lowering. The Oslo study of diet and smoking intervention was conducted in 1,232 healthy normotensive men ages 40 to 49 years at risk for CHD with cholesterol levels of 290-380 mg/dL (7.50-9.83 mmol/L).[330] Mean blood cholesterol levels were approximately 13 percent lower in the intervention group than in the control group during the trial. The mean tobacco consumption per man decreased by 45 percent more in the intervention group. The 5-year incidence of acute myocardial infarction was reduced by almost 50 percent in the intervention group. Statistical analysis suggested that the reduction in the incidence of myocardial infarction was correlated with the reduction in total cholesterol levels and, to a lesser extent, with smoking reduction.

The Multiple Risk Factor Intervention Trial was a randomized primary prevention trial of 12,866 high-risk men ages 35 to 37.[331] The special intervention (SI) group received treatment for hypertension, counseling for cigarette smoking, and dietary advice for lowering blood cholesterol. Despite an overall improvement in their risk factor status, CHD mortality of the men in the SI groups was reduced by only 7.1 percent compared with that in the usual care (UC) group. Analysis of blood cholesterol data from the SI group showed a moderate reduction in their average level over the course of the study. However, a reduction also occurred in the UC group so that the SI-UC difference amounted to only 4-6 mg/dL (0.10-0.16 mmol/L), or 2 percent, about half of the original goal. Thus the ability of MRFIT to evaluate the benefit of cholesterol lowering was compromised. Analyses of a subgroup of the MRFIT participants that resembled the Oslo study participants suggested that diet and smoking interventions may have been beneficial: within the SI group there was a statistically significant reduction in the risk of CHD death in those who both had a reduced cholesterol level and reduced smoking.[332] A 10 mg/dL (0.26 mmol/L) reduction in cholesterol was estimated to be associated with a 6.6 percent reduction in the CHD death rate.

Peto and colleagues,[179] using a meta-analysis technique, evaluated 18 published and 2 unpublished randomized trials of cholesterol lowering by diet and/or drugs, as described previously. Analysis of all data from these trials indicates that a 10 percent reduction in cholesterol was associated with an average reduction of 16 percent in CHD risk in trials lasting 4 years. Peto also noted that the reduction in CHD risk varied according to the duration of treatment: an average of 11 percent in 13 shorter trials and 21 percent in 7 longer trials, for a 10 percent reduction in blood total cholesterol. The findings in clinical trials are consistent with those of observational epidemiologic studies.[333] There have been no trials lasting a decade or longer. Peto concludes that results from the observational studies suggest that a reduction of 10 percent in total cholesterol over decades would be associated with a 33 percent reduction in rate of fatal CHD. In a similar overview of 22 randomized trials of cholesterol lowering, Yusuf et al.[334] found significant reductions in coronary mortality and morbidity. For a standardized reduction in cholesterol level of a fixed duration, there was no apparent heterogeneity in the reduction of CHD observed in the drug or dietary intervention trials.

Although cholesterol-lowering clinical trials have provided conclusive evidence that both nonfatal and fatal coronary events are significantly reduced, overall mortality has tended to remain unchanged. It has been difficult to draw sound conclusions regarding this issue, especially given the small numbers of events that are usually involved, and the lack of statistical power in the experimental design of many trials to detect changes in non-CHD mortality. In those studies in which noncoronary mortality has tended to increase, little consistency has been observed in the various causes of noncoronary death, which have included diverse accidents and violent deaths (homicides, suicides, road accidents), cancer, and miscellaneous other medical causes of death. In the World Health Organization study of clofibrate therapy, overall mortality was increased.[335] The question of whether an excessively low blood cholesterol carries adverse health risk is further considered later in this report.

Some reassurance has been gained from the results of the niacin-treated subgroup of the Coronary Drug Project. The Coronary Drug Project, a long-term study of lipid-influencing drugs in male survivors of myocardial infarction, was concluded in early 1975.[336] At that time niacin treatment showed benefit in decreasing definite nonfatal recurrent myocardial infarction but did not decrease total mortality. Later analyses, with a mean followup of 15 years, nearly 9 years after termination of the trial, showed that mortality from all causes in the niacin group was 11 percent lower than in the placebo group (52.0 versus 58.2 percent; $p=0.0004$).[337] This late benefit of niacin, occurring after discontinuation of the drug, is likely to be the result of a translation into mortality benefit over subsequent years of the early favorable effect of niacin in altering lipoproteins and decreasing the rate of recurrence of nonfatal myocardial infarction. In another trial of pharmacologic reduction of blood cholesterol in patients after myocardial infarction, Carlson and Rosenhamer[338] reported significant reductions of both cholesterol and triglycerides in the group receiving clofibrate and nicotinic acid. In this Stockholm Ischemic Heart Disease Secondary Prevention Study, total mortality was significantly reduced, and CHD mortality was reduced to an even greater degree. In addition, the Oslo Study Diet and Antismoking Trial[339] has reported results after 102 months, describing significant reductions in sudden death and total coronary events, with marginally significant reductions in total mortality.

Any single clinical trial has design features specific to it that limit its extrapolation. However, the broad consistency of the results of the many major cholesterol-lowering studies appears to have general implications. The cholesterol-lowering trials were designed to test the hypothesis that reducing elevated cholesterol levels would reduce the risk of CHD. Most studies support this hypothesis, indicating that reduction of blood

cholesterol has a beneficial effect on CHD irrespective of whether it is produced by cholesterol-lowering drugs or by dietary means. These benefits are found in individuals with or without prior manifestation of CHD. The findings also suggest that benefits are seen whether pretreatment cholesterol levels are especially high or only moderately elevated. Finally, the greater the cholesterol reduction, the greater the reduction in risk.

It was pointed out earlier that it is highly unlikely that an entirely satisfactory primary prevention study of diet-induced cholesterol lowering to reduce CHD can or will be conducted. In the absence of such a study it is, however, reassuring to note that the diet-induced cholesterol-lowering trials also appear to have reduced the risk of clinical CHD in proportion to the amount of cholesterol lowering that they have achieved, and have also been associated with a decreased rate of coronary lesion progression in angiographic studies.[340] When dietary change has been studied together with other interventions, the results have been more variable, but this may depend on the amount of cholesterol lowering achieved in these studies.

On the basis of the experience of the clinical trials, a rule of thumb has been generated, namely that for each 1 percent reduction in blood cholesterol, an approximate 2 percent reduction in CHD incidence can be expected. This relationship has considerable public health and clinical implications.

b. Community Trials

Epidemiologic studies have long demonstrated the strong relationship between culture, environment, diet, and cardiovascular disease.[341] Food patterns are strongly rooted in cultural, agricultural, and economic factors. Hence community-wide changes in eating patterns may be necessary to produce population changes in disease rates. In fact, Stamler[342] suggests that the recent decline in CHD found in many industrialized countries may, to a significant degree, be the result of national shifts in food composition and consumption patterns as well as control of other risk factors. These observations have led to a number of experiments to test approaches to educating entire populations on dietary factors that might contribute to lower blood cholesterol levels. While these community studies involve entire populations, most are experimental in that comparison populations are studied to serve as a reference for ascertaining community trends.

Multiple educational approaches have been used to attain the goals of these community programs. Prevention of CHD—both through promotion of healthy eating patterns and through detection and treatment of those with high blood cholesterol levels—has been one important goal. Some studies have used the health care system both to detect high-risk individuals and to educate patient groups. Others have attempted to influence food production and delivery by altering recipes and labeling food products to meet low-cholesterol and low-fat specifications. Mass cholesterol screening programs have been started and tested along with community education programs involving both direct and mass media approaches. All have a goal of testing whether CHD can be reduced in entire populations.

Noteworthy among the several early studies of community programs is the North Karelia study, which began in the early 1970's. This project was a response to observational studies that had found very high blood cholesterol levels and CHD rates in this part of Finland. Ten-year followup studies demonstrated that the average blood cholesterol level was 3 percent lower in the county in which a cholesterol education program had been conducted, compared with a reference area,[343-345] and that this was associated with declining CHD rates. The Stanford Three-Community Study, a project that used a mass media approach, demonstrated lower cholesterol levels in the adult population of the study communities compared with the reference town.[346] Studies in Switzerland[347] and Israel[348] likewise demonstrated lower cholesterol levels in the study community compared with reference areas. A Polish study was less successful, showing little cholesterol change, although other CHD risk factors were improved.[349]

There are also several ongoing community studies attempting to lower population distributions of cholesterol. They all aim at both high-risk individuals and the general population. The Stanford experience has been extended to a larger five-city study.[350] Preliminary results show that cholesterol has been lowered an average of 6.0 mg/dL (0.16 mmol/L) in the two study communities.[351] In Minnesota, large-scale systematic screening programs supported by multiple community education strategies have shown significant lowering of cholesterol (6.6 mg/dL [0.17 mmol/L]) in study communities compared with reference populations.[352] In Pawtucket, Rhode Island, a similar screening approach recruiting volunteers has shown significant reduction in cholesterol levels at followup.[353]

Each of these studies has used different strategies to effect dietary change across the population to produce blood cholesterol reduction. Data from these studies also suggest that blood cholesterol reduction can be achieved in people across the entire cholesterol distribution rather than just in those with very high blood cholesterol levels.

6. Other Risk Factors for CHD

Other risk factors for CHD fall into two broad categories: "nonmodifiable" or "modifiable." Increasing age, male sex, and family history of premature CHD are the three major nonmodifiable risk factors.[163,354] The three major modifiable risk factors are elevated blood cholesterol, high blood pressure, and cigarette smoking.[151,165,180,206,355-357] Other significant modifiable risk factors include physical inactivity and obesity.[155,227,358-360] A low level of HDL-cholesterol also increases CHD risk. The level of HDL-cholesterol, although less modifiable than the level of total or LDL-cholesterol, can often be altered by changes in smoking practice, body weight, exercise, and, to a limited extent, by changes in fatty acid intake. Diabetes mellitus is another risk factor, but the effectiveness of therapy in offsetting the increase in CHD risk due to diabetes is uncertain. These various risk factors appear to act in concert to increase risk with their effects at least additive and in some cases multiplicative.

The three major modifiable risk factors for CHD contribute to the development of a large proportion of cases of CHD. As the level of each major risk factor rises, so too does the risk of developing CHD. For example, a blood pressure level of 160/95 mm Hg increases risk twofold compared with a level below 140/90 mm Hg, and a level of 200/120 mm Hg increases CHD risk fivefold, at any given cholesterol level. When all three risk factors are present to a moderate degree—for example, blood cholesterol of 240 mg/dL (6.21 mmol/L), blood pressure of 160/95 mm Hg, and one-pack-per-day cigarette smoking—the risk increases manyfold. When the levels of these risk factors are higher, the resultant multiplicative risk increases proportionately, rising as high as twentyfold.

Almost one-third of all adults in the United States have high blood pressure, defined either as having a systolic blood pressure $\geq$140 mm Hg and/or a diastolic blood pressure $\geq$90 mm Hg, or as being on antihypertensive therapy.[361] By age 65, more than 60 percent of U.S. adults have undesirably elevated blood pressure. Nonpharmacologic measures to produce healthful lifestyle habits are now recommended as the first line of therapy for high blood pressure.[361] Such measures include reducing dietary consumption of sodium chloride and fat, maintaining desirable body weight, moderating alcohol intake, and being physically active.

For many individuals, pharmacologic agents are also necessary in order to achieve the goal of maintaining blood pressure <140/90 mm Hg. However, some pharmacologic agents used to treat high blood pressure have adverse effects on serum lipoprotein levels.[362,363] Such effects include increases in the levels of total cholesterol, LDL-cholesterol, and triglycerides, and decreases in the levels of HDL-cholesterol. These effects can be reduced or even prevented by modifying the quantity and composition of dietary fat intake, by selecting drugs without these effects, or by limiting the use of drugs with undesirable effects on lipids.

The risk of developing CHD is also strongly influenced by cigarette smoking. The more cigarettes an individual smokes each day, the higher the risk. This increased risk from smoking holds true for both filtered and nonfiltered cigarettes.[364] Anyone who smokes one pack a day has almost twice the CHD risk of a nonsmoker, and anyone smoking two packs a day has three times the nonsmoker's risk. Cigarette smoking is consistently associated with lower HDL-cholesterol levels.[365] Smoking cessation is associated with increases in HDL-cholesterol levels.[366] Smoking also increases the risk of developing at least eight types of cancer as well as the risk of a stroke.[367,368] Recommendations to all people who smoke to quit are based on very solid evidence.

The most prevalent of the other modifiable risk factors for CHD is physical inactivity. As with the major risk factors, the risk is graded in nature; that is, the more inactive a person is, the greater the risk. Conversely, the more active a person habitually is, the lower the risk.[369] On the average, physical inactivity increases the risk of developing CHD by one and one-half to two times. Physical inactivity is associated with lower levels of HDL-cholesterol.[370] Inactivity also is strongly associated with excess weight, which frequently results in increases in the levels of total cholesterol, LDL-cholesterol, blood sugar, and blood pressure.[371]

Presence of obesity,[372,373] defined as a body mass index (weight in kilograms/height in meters squared) of greater than 30, is a major concern. Four studies based upon life insurance data carried out between 1903 and 1979 have identified a quadratic relationship between relative weight and mortality, with the highest mortality ratios being found in those who were extremely obese. For instance, the Build Study[374] demonstrated a steep, curvilinear relationship between relative weight at entry and subsequent mortality among men age 15 to 39. This relationship is less striking for those in older age groups, which raises the question of whether weight control is more important in terms of overall morbidity and mortality for younger individuals than for those over the age of 50 years.

The relationship of obesity to CHD is complex. The Framingham study originally observed that obesity was predictive of the development of CHD and was also closely related to levels of blood total cholesterol, blood pressure, blood sugar, and uric acid. However, when obesity was included in an analytic model that included these other variables, it did not make an independent contribution to risk. After 26 years of followup of the original Framingham cohort, Hubert et al.,[227] however, reported that obesity did make an independent contribution to estimating the risk of cardiovascular disease. They also observed that obesity did not exert its effect exclusively through its association with serum lipids, blood pressure, and blood sugar.

Some other prospective studies of cardiovascular diseases have not been able to show such a clear relationship. For instance, the data from eight cohort studies of the U.S. Pooling Project were inconsistent—some showed no relationship, others a U-shaped association, and only one confirmed the findings of the Framingham Study indicating an independent risk factor status of obesity. However, the pooled data did reveal a positive relationship.[151]

The most powerful effect of obesity on both cardiovascular morbidity and mortality is probably due to its effect on other cardiovascular risk factors. Not only does obesity increase blood pressure, blood sugar, and the risk of noninsulin-dependent diabetes mellitus (NIDDM), but it is associated with an increase in the LDL-cholesterol levels, a decrease in the HDL-cholesterol levels, and an increase in the fasting triglyceride levels.[249,375] There is a consistent correlation between body mass index and total blood cholesterol level.[376]

A low level of HDL-cholesterol has been linked to an increased risk of developing CHD, particularly in persons over age 50.[196] Conversely, a high level of HDL-cholesterol is associated with a reduced CHD risk. HDL-cholesterol levels <35 mg/dL (<0.91 mmol/L) are associated with four times the risk compared with levels >75 mg/dL (>1.94 mmol/L). Accordingly, a level of HDL-cholesterol below 35 mg/dL (0.91 mmol/L) has been classified as a major CHD risk factor by the Adult

Treatment Panel of the NCEP.[3] HDL-cholesterol levels tend to be higher in women than in men and in individuals who are physically active, do not smoke, and are lean. An association of high HDL levels with alcohol intake is also recognized, but it is unclear whether the net effect on health is beneficial even at low levels of intake. On the other hand, the undesirable social, economic, and health consequences of high alcohol intakes are well recognized. Accordingly, alcohol has not been recommended as a means of raising HDL-cholesterol.[377]

Diabetes mellitus substantially increases the risk of developing CHD both in men and in women.[359] This increased risk holds true for both insulin-dependent diabetes and for NIDDM.

There are approximately 5 million known people with diabetes mellitus in the United States today. It is estimated that there are an additional 5 million individuals with diabetes mellitus who have not yet been diagnosed. More than 85 percent of diabetic persons have NIDDM, the major precipitating cause of which is excess weight.

As with nondiabetic individuals, the presence of blood lipid abnormalities in diabetic people increases the risk of developing CHD and of stroke.[378] However, the incidence of these abnormalities is increased in poorly controlled diabetes.[379] Specifically, blood levels of triglycerides, cholesterol, VLDL-cholesterol, and LDL-cholesterol all tend to be higher and HDL-cholesterol tends to be lower with poor glycemic control. When glycemic control improves, the level of HDL-cholesterol usually rises, and the levels of the other lipids and lipoproteins usually fall. Because of the influence of glycemic control on lipid levels, it is most appropriate to measure lipids in diabetic people after good glycemic control has been achieved.

In the past, it was generally recommended that diabetic persons follow a diet relatively high in protein and fat. It was thought that a relatively low carbohydrate diet would improve the degree of glycemic control. However, because of the high incidence of CHD in diabetic individuals[380] and because of new evidence concerning the beneficial effects of increased carbohydrate and fiber on glycemic control, dietary recommendations have recently been significantly modified by the American Diabetes Association (ADA) nutrition task force.[381] The revised recommendations are similar to those of the ATP report. Specifically, all diabetic persons are urged to follow a diet with about 30 percent of the calories from fat, with less than 10 percent of those calories to come from SFA. For those individuals whose blood cholesterol levels remain high, the ADA recommends that SFA be reduced even further.

The American Diabetes Association recommends that obese diabetic persons lose excess weight and seek to maintain their desirable weight, thereby often correcting high levels of blood cholesterol, blood pressure, and their underlying diabetes. Finally, recommendations have recently been made to have diabetic persons increase their fiber consumption substantially, in an effort both to reduce blood cholesterol levels and to improve glycemic control.[382]

In summary, there are numerous risk factors in addition to elevated blood cholesterol that play an important role in the development of CHD.

7. Low Blood Cholesterol and Risk

An important question is whether there are any inherent undesirable health effects that may come from an excessive lowering of blood cholesterol levels. Concern over a possible relationship between low blood cholesterol levels and risk of cancer was raised by the report of Pearce and Dayton.[383] Their clinical trial of the effects of a diet high in polyunsaturated fat on blood cholesterol showed more carcinoma deaths in the diet groups. Shortly after publication of those results, an analysis of five clinical trials relating to cholesterol lowering was published.[384] The conclusion reached was that cholesterol-lowering diets do not influence cancer risk, but the need for more data was expressed.

Since 1971, there have been a number of publications concerning low blood cholesterol and cancer risk.[385,386] McMichael et al.[387] reviewed the data available through 1983. An inverse relationship between blood cholesterol levels and cancer risk was found in 12 studies, but not in 8 others. All the reports cited had originally been studies of cardiovascular disease.

The general characteristics of these studies were that they all related to men. The cancer most often observed in these studies was colon cancer. Several of the studies showed an inverse relationship that did not attain statistical significance. One study[388] found a significant inverse relationship with total cancer mortality. In about half the studies, the inverse relation between blood cholesterol and cancer was observed during the early followup period, suggesting that the low cholesterol levels may have been due to preclinical effects of cancer. However, in a few studies the association with low cholesterol persisted for 10 years. This evidence has prompted the suggestion that males with naturally occurring low blood cholesterol may be at increased risk of colon cancer.

Since 1984, several studies concerning the relationship between blood cholesterol and noncardiovascular disease risk have been reported. Martin et al.[165] presented a graph in which age-adjusted 6-year total mortality and CHD mortality were plotted against serum cholesterol for the 361,662 men screened for the MRFIT program. The CHD mortality curve rose continuously from 140 mg/dL (3.62 mmol/L) of serum cholesterol. The curve representing total mortality rates showed the lowest rates for an average cholesterol level between 165 mg/dL (4.27 mmol/L) and 185 mg/dL (4.78 mmol/L). A small increase in total mortality rate occurred in the men with cholesterol levels below 165 mg/dL (4.27 mmol/L) although the CHD death rate was lower. Ten-year followup data on this group of men show similar results.[1] Schatzkin et al.[389,390] have analyzed blood cholesterol in the NHANES I followup, using data derived from a cohort of 5,125 men and 7,363 women. Among men, cancer (all site) morbidity and mortality was somewhat higher in the bottom two quintiles of cholesterol, namely 183-205 mg/dL (4.73-5.30 mmol/L) and below 183 mg/dL (4.73 mmol/L). In women, cancer mortality and morbidity was higher only in those groups whose cholesterol levels were below 179 mg/dL (4.63 mmol/L). Wingard et al.[391] found no relation between cholesterol levels and cancer morbidity or mortality in 4,035 Californians. Hiatt and Fireman[392] examined data on more than 160,000 men and women in a California health plan. Their conclusion was that low blood cholesterol did not increase the risk of cancer but that preclinical cancer reduced blood cholesterol levels. Mannes et al.[393] have actually suggested a small positive association between blood cholesterol and colorectal cancer. Tornberg et al.[394] have found a similar association. On the other hand, Salmond et al.,[395] in reporting on 630 New Zealand Maori followed over 17 years, found significant inverse relationships between cholesterol and either cancer or all-cause mortality in men and women and suggest that the association may not be due to undetected illness. Neither the LRC-CPPT[323] nor the Helsinki Heart Study[325] showed an increase in incident cancer causes in treatment compared to control groups. Overall, the evidence available indicates that dietary change to lower blood cholesterol is highly unlikely to increase the risk of colon cancer; indeed, there is some epidemiologic evidence suggesting that a habitual diet high in fat and SFA (which would be expected to raise the blood cholesterol level) increases the risk of colon cancer.[5]

Several studies suggest that hemorrhagic stroke occurs more often in population groups with low levels of blood cholesterol. Hemorrhagic stroke is less common in Americans than in some other groups.[396,397] The relationship between hemorrhagic stroke and low blood cholesterol has been noted in people of Japanese heritage both in Japan[398] and in Hawaii.[399] A similar relationship has been noted among women in the Framingham Study,[156] and in the men screened for the MRFIT study.[400] MRFIT results are particularly noteworthy. There was a statistically significant increased likelihood of hemorrhagic stroke, especially intracranial, in men in the lowest quintile (<160 mg/dL or <4.14 mmol/L) of total blood cholesterol. Overall, men with nonhemorrhagic strokes had a significantly higher average blood cholesterol level than did those who experienced no stroke during the 6 years of followup. The average blood cholesterol levels of those with subarachnoid or intracranial hemorrhage were slightly, but not significantly, lower than those who had no stroke. Men with any of the types of cerebral lesions were more likely to be smokers and to have elevated blood pressure. Given that the age-adjusted death rates for CHD in the MRFIT screenees were 60.5 per 10,000 men, compared with 5.48 for nonhemorrhagic stroke and only 2.36 for intracranial hemorrhage, and given the clear curvilinear relationship between CHD and serum cholesterol levels (figure 1, page 7), Iso and colleagues[400] conclude that the small risk of a low blood cholesterol level in American men is far outweighed by the risks from CHD and nonhemorrhagic stroke associated with even mild elevations of blood cholesterol.

The benefit-risk relationship is important in any public health initiative. While the relationships among diet, blood cholesterol, and disease require further research, the data suggest that any additional risk of blood cholesterol lowering is either nonexistent or very small. The issue of potential competing risks and benefits associated with dietary changes has been discussed in detail in the report *Diet and Health: Implications for Reducing Chronic Disease Risk.*[5] That report concludes that the public health benefits that will result from dietary changes to lower blood cholesterol greatly exceed any small adverse effects that might also occur.

B. Eating Patterns in the United States

1. Current Dietary Patterns in the United States

In September 1989, the ad hoc Expert Panel on Nutrition Monitoring (EPONM), a committee established at the request of the U.S. Department of Agriculture and the U.S. Department of Health and Human Services, issued a report containing current data on the nutritional status of the United States population.[401] The EPONM concluded that the principal nutrition-related health problems in the United States are associated with excess consumption of food energy (calories), fat, saturated fatty acids, cholesterol, sodium, and alcohol. The report also stated that one-fourth of the adults in the United States are overweight.[401] Planning for achievement of the nutrition objectives for the nation are consistent with these nutrition-related problems.[402]

The EPONM report noted that certain subgroups within the U.S. population had low intakes of iron and vitamin C, including young children and females of childbearing age, especially those who were black, poor, or both. Concern was also expressed that calcium intakes of young women may be inadequate for the development of maximal bone mass. In addition, intakes of zinc, vitamin B_6, and folacin were lower than recommended levels in some groups, especially women.

2. Fat in the American Diet

The Continuing Survey of Food Intakes by Individuals[14-17] showed that the fat intake by men and women 19 to 50 years of age averaged 36-37 percent of total energy intake. Using a "food group" approach which classifies foods by their major ingredients, about 50-55 percent of the fat and 55-60 percent of the SFA was estimated to come from meat, poultry, fish, egg and dairy products, and from mixed dishes containing these animal products as major ingredients.[403] These estimates include fats of other origins such as frying fats and salad dressings used in preparation of these foods. The estimates exclude the fat and SFA from the animal products in a mixed dish included in another food group, for example the meat and cheese included in spaghetti or pizza. An "ingredient approach" that separates foods into their ingredients has also been used to estimate sources of fat, although the analysis is limited to women's diets. These preliminary data show that about 50 percent of the fat and about 59 percent of SFA came from meat, poultry, fish, egg, and dairy products. The ingredient approach also showed that fats and oils added at the table or in food processing and preparation accounted for at least 33 percent of the fat in women's diets.[404]

The palm, palm kernel, and coconut oils (so-called "tropical oils") contain as high or higher proportions of SFA's than animal fats.[11] In 1985 these tropical oils provided about 3 percent of the total amount of fat in the U.S. food supply.[403] Current intakes of the three tropical oils combined are estimated to be about 2 to 4 grams per person per day. These estimates represent less than 4 percent of total fat intake, less than 2 percent of the daily energy intake, and 8 percent or less of the SFA intake by the U.S. population.[403]

In the recommendations for dietary change to lower blood cholesterol level, much of the positive contribution of animal products to vitamin and mineral nutritional status can be maintained. This can be achieved by placing emphasis on lean and lower-fat animal product choices and by limiting portion sizes, rather than avoidance or elimination of foods of animal origin.[405]

3. Change Required in American Diet To Achieve Various Targets

Current dietary intakes of Americans have been compared to those recommended by the American Heart Association's Step-One diet (which is identical to the Step-One diet of the NCEP Adult Treatment Panel).[3,406] If in fact the entire population changed its eating patterns to meet the AHA / NCEP Step-One recommendations, intakes of the target nutrients, total fat, and saturated fatty acids would have to be altered significantly downward from current consumption levels.[407] It is likely that such changes are feasible in the future, given technological improvements in food processing and preparation that facilitate lowering dietary fat and SFA while maintaining acceptable palatability and nutritional adequacy.

4. Dietary Safety and Adequacy

Hansen and Windham[407] have expressed concern that if fat and cholesterol were decreased to levels recommended in the Step-One diet, deficiencies might occur in certain "problem" nutrients. These nutrients are regarded as problematic because intakes are already low in American diets in comparison with nutrient standards such as the recommended dietary allowances (RDA) of the National Academy of Sciences.[265] These problem nutrients include zinc, iron, calcium, and folacin (folic acid). The proportion of the population consuming less than the RDA for iron and folacin will be reduced substantially with the reduction in several of the relevant RDA's in the 1989 recommendations. Choosing low-fat animal foods permits utilization of the highly bioavailable sources of those nutrients in animal foods. Adequate attention to nutrition education for the public will help ensure achievement of RDA levels including those for zinc, iron, calcium, and folacin. Overall, there is extensive epidemiologic evidence that low-fat diets are not harmful to health; this has been carefully reviewed recently.[265]

Menus for adults that meet the RDA's for all nutrients while meeting low-fat, low-SFA, and low-cholesterol targets can be created by helping individuals make informed food choices. For example, Dougherty et al.[405] found, with diets planned and served in a metabolic ward, that it was possible to improve intakes of vitamins and minerals while decreasing SFA, total fat, and cholesterol intake to target levels. Usual intakes of meats, milk products, fish, and eggs were sustained. Intakes of vitamins B_1, B_2, B_6, B_{12}, and C increased, as did potassium, calcium, magnesium, phosphorus, zinc, and iron. Eating patterns designed to lower blood cholesterol can be as high or higher in nutritional quality as other diets.[11,265]

This also applies to diets of children and adolescents. The feasibility of such menu planning for children and adolescents, with their increased needs for nutrients per calorie, has also been demonstrated previously using the 30 percent fat, 10 percent SFA, and low dietary cholesterol specifications.[408] Moreover, observational studies indicate that even children who are vegetarian and consume diets very low in fat and cholesterol can achieve and maintain good nutritional status, whereas those who are fed nutritionally unplanned diets are the individuals who may fall into difficulties.[409]

5. Special Concerns Involving Women

Although the nutrient densities of men's and women's diets are similar, the considerably smaller energy intakes of women, coupled with increased requirements for some essential nutrients such as iron and calcium, highlight the need for guidance for women to ensure nutritional adequacy. This becomes even more important with the reduced energy intakes that occur as women become older. Information on the reported dietary intake of women, available from the USDA Continuing Survey of Food Intakes by Individuals (CSFII) is shown in table 5, page 68.[14,15] For women ages 19-50 years, the percentage of calories from fat was 37 percent. The corresponding percentage distribution of calories from fat was as follows: SFA, 13; MUFA, 14; and PUFA, 7. Only 10 percent of women ages 19-50 years were below the level of 10 percent of calories from SFA. About 65 percent of women attained a level of 10 percent or more of calories from MUFA, while 86 percent of women consumed less than 10 percent of calories from PUFA. About 62 percent of women ages 19-50 years had diets that provided 300 mg or less of cholesterol per day.

As previously described, the EPONM[401] concluded that women tend to have a lower calcium intake than recommended and that intakes of iron and vitamin C do not meet RDA's for some women of childbearing age. According to the 1985 USDA CSFII data–for calcium–78 percent of women did not meet 100 percent of the RDA; and 54 percent did not achieve 70 percent of the RDA; several other nutrients—including B_6 and folacin—were below recommended levels in diets of many women. The new RDA standards for folacin and iron will mean that more women meet these standards.[265]

According to NHANES II,[2] more than 28 percent of U.S. adult women have blood cholesterol levels at or over 240 mg/dL (6.21 mmol/L). More than 58 percent of women have blood cholesterol levels that are above 200 mg/dL (5.17 mmol/L). These data on the prevalence of high blood cholesterol and data on the nutrient intakes of women, particularly adolescents and women of childbearing age,[412] emphasize the importance of developing detailed dietary recommendations to lower blood cholesterol levels in women. Cleveland[413] used a computer-generated model to estimate changes in women's reported intakes to meet a target diet containing 1,600 calories, <30 percent of calories from fat, <10 percent of calories from saturated fatty acids, <300 mg/day cholesterol, over 50 percent of calories from carbohydrates, 3,300 mg/day sodium, 20 g/day fiber, and 100 percent RDA for vitamins and minerals. Within major food groups, the model relied on lower-fat, leaner choices. A pattern meeting the specified nutritional criteria contained at least twice as much low-fat milk, lean red meat, legumes, whole-grain bread, cereal, and vegetables as the estimated usual consumption pattern. It contained almost no bacon, sausage or lunchmeat, cheese, grain-based snacks, desserts, fats added at the table, or sugars and candies.

C. What Influences Eating Patterns?

1. Overview

Various factors influence eating patterns and ultimately many aspects of health.[414] These factors fall into four major groups: *food composition* (e.g., genetics, manufacturing); *food supply* (production, processing, distribution, cost, and many other factors affecting accessibility); *food demand* (income, relative price structures of food and other needs, education, cultural influences, food habits, food taste, taxes, advertising, public relations and consumption stimulation, programs sponsored by government or special interest groups); and *nutritional needs* (factors influencing either the individual's state of health or biological utilization of food). Figure 7, page 70, lists many of the factors that influence food consumption, and the pages that follow describe much of the existing research on these factors. Some, but not all, of these factors have been carefully studied, and some have been subjects of intervention research. Other factors, such as new methods of distributing, preparing, or cooking food, have not been carefully analyzed,

Table 5

Mean Daily Intake[a] of Food Energy, Nutrients, and Food Components for Men, Women, and Young Children from the Continuing Survey of Food Intakes by Individuals (CSFII), 1985[b]*

	Men	**Women**	**Children**
Total Food Energy (% REI)[c]	(94)	(82)	(100)
Fat [% total energy]			
Total fat	[36]	[37]	[34]
Saturated fatty acids	[13]	[13]	[14]
Monounsaturated fatty acids	[14]	[14]	[12]
Polyunsaturated fatty acids	[7]	[7]	[6]
Cholesterol mg	435	304	254
Protein			
[% total energy]	[16]	[16]	[16]
(% RDA)[d]	(175)	(144)	(222)
Carbohydrates [% total energy]	[45]	[46]	[52]
Dietary Fiber g	18	12	10
Vitamins (% RDA)			
Vitamin A	(122)	(127)	(215)
Vitamin E	(98)	(97)	(108)
Vitamin C	(182)	(133)	(186)
Thiamin	(124)	(110)	(153)
Riboflavin	(129)	(115)	(197)
Niacin	(146)	(130)	(151)
Vitamin B_6	(85)	(61)	(127)
Vitamin B_{12}	(245)	(156)	(192)
Folacin	(76)[f]	(51)[f]	(157)[f]
Minerals (% RDA)			
Calcium	(115)	(78)	(105)
Phosphorus	(192)	(126)	(132)
Iron	(159)	(61)[f]	(88)[f]
Zinc	(94)[f]	(60)	(84)
Magnesium	(94)	(72)	(121)
Minerals (ESADDI)[e]			
Sodium	(exceeds)	(within)	(exceeds)
Potassium	(within)	(within)	(1-3 years exceeds) (4-5 years within)
Copper	(below)	(below)	(below)

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* The Surgeon General's Report on Nutrition and Health. U.S. Department of Health and Human Services. Public Health Service USDHHS (PHS)Publication No. 88-50210, 1988.[410]

[a] Estimated mean daily intake is expressed in several ways: amount of intake, percent of total energy intake, percent of 1980 Recommended Dietary Allowance, or comparison with Estimated Safe and Adequate Daily Dietary Intake.

[b] Data based on 1-day dietary recalls obtained by personal interview for 658 men 19 to 50 years of age, for 1,459 women 19 to 50 years of age, and for 489 of their children 1 to 5 years of age in 1985 (unweighted numbers). Nutrient intakes do not include vitamin and mineral supplements or sodium from salt added at the table.

[c] Recommended Energy Intake;[411] Source of percentages: NFCS, CSFII Report Nos. 85-1 and 85-3.[14,15]

[d] Recommended Dietary Allowance;[411] Source of percentages: NFCS, CSFII Report Nos. 85-1 and 85-3.[14,15]

[e] Estimated Safe and Adequate Daily Dietary Intake.[411]

[f] Figures given are based upon 1980 RDA's; the 1989 RDA's for these nutrients are substantially lower.

even though some of them, such as microwave cooking, have potentially major effects.

2. Factors That Influence Eating Patterns and Approaches to Changing Them

a. Food Supply

Food producers and processors influence the food supply in a variety of ways, which are briefly considered in the following section. Many of these food supply factors are amenable to change by societal or industry-wide efforts but are less influenced by individual efforts.

- Food Production, Processing, Distribution, and Marketing

  The production, processing, distribution, and marketing of food influences eating patterns in ways that may either promote or detract from cholesterol-lowering efforts.[415] *Designing Foods*[11] describes some of the many barriers that hinder the animal agriculture industry from achieving a food supply that is lower in fat and cholesterol and suggests ways of overcoming these barriers.

- Consumer Information on Foods

  The American food supply is one of the most abundant, varied, and economical in the world. With the average supermarket selling more than 24,000 items, the consumer is faced with a myriad of food choices. For individuals who wish to alter the type and amount of fat and the amount of cholesterol in their diets, some of these food choices are more appropriate than others.[416] The problem is not that suitable foods are unavailable, but that it is difficult while food shopping to know which choices are best.[417] Increased access to information to guide food choices can help.[418] The knowledge, skills, and behavior of those who prepare foods is also important.

- Ecosystem

  On a worldwide basis, the ecosystem—as determined by geography, climate, labor, capital, and other factors—is critical in shaping the agricultural base and the food supply. It is a potent and changing influence on food availability and thus on eating patterns.

b. Societal Factors

Influences on eating behavior are of two different kinds: those that operate at the societal level, over which individuals have relatively little influence, and those at the personal level, over which individuals exert more control. This review emphasizes factors subject to individual control in addition to societal policies and practices that affect eating and lifestyle behaviors. This emphasis is not because changes at the societal level are unimportant, but because, in the short run, changes are more likely to occur first at the individual level. Collectively, such changes at the individual level foster society-wide change since they influence demand and strengthen the political will that is necessary to change policies and regulations in the public, private, and voluntary sectors. Eating patterns are also affected by policies related to the food chain (food production, agriculture, food science, economics, food manufacturing or distribution) and to health care, as well as to broader economic and educational factors.

- Cultural Factors

  Culture is a major influence on food selection.[419-421] Culture influences food-related behavior—the ways in which people select, consume, and use the available food supply in response to social and cultural pressures.[420,422] A great deal of intra- and interindividual variability exists in eating habits even in this country, which has a relatively consistent national food supply.[423] In a given society, there are subcultures that have very different norms and values, which may influence the effectiveness of health-related interventions (see section VI, Ethnic, Cultural, and Minority Characteristics That Influence Diet and Health).

Figure 7

Eating Pattern Determinants

The boxes depict the major elements in the food chain from the food science base to the outcomes of food consumption. Major influences on the food chain, each with an impact on several of the food chain steps, are listed.

Food Science Base	Food Manufacture	Food Distribution	Food Purchasing	Food Preparation	Food Consumption	Food Consumption Outcomes
Nutrition Biochemistry Preservation Genetics	Agriculture Synthesis Processing Additives Modifiers Hybridizers Mass Preparation	Wholesale Retail Product-Specific Route-Specific	Cost Culture Advertising Knowledge Health	In-House Restaurants Institutions	Socialization Education Nutritive Value Health Culture Taste Cost Mood	Pleasure Health Deficiencies Surpluses

Major influences on the food chain:

Agribusiness
Conglomerates
Grocery Chains
Advertising
Profitability
Special Interest Groups
Media

Government Agencies
Health Professionals
General Education
Nutrition Education
Food Science Education
Culinary Education

- Other Societal Influences

A wide variety of other societal factors influence food demand. Economic factors include stimulation of consumption, rationing, and differential taxes (such as taxes on alcohol and cigarettes). These and similar measures have attempted to alter demand for food, sometimes in directions counter to an objective of lowering blood cholesterol levels. Some promotional efforts to stimulate consumption are viewed as successful enough to be supported on a continuing basis by producers. Examples include programs of the National Dairy Research and Promotion Board, the Egg Board, the Beef Check Off Program, the National Dairy Council's Bridge Project, and some programs administered by the Government with contributions by producers. Several of these have emphasized lower fat and lower cholesterol consumption options.

- Political Factors

  Historically, many attempts have been made to influence what people eat by changing policies, laws, and regulations. More recently, public health research and practice have turned toward efforts to modify eating behavior by modifying the environment within institutions and communities. If the political will is present for making changes, such measures can be very effective, as recent smoking-related interventions have shown.

  Political factors can both hinder and foster healthful changes in food consumption. In making nutrition policy, political, social, and economic considerations must be weighed along with the science base. Therefore, it is important to distinguish clearly between advice based on scientifically established evidence, deductions from indirect evidence, and conjectures based on assumptions, no matter how logical they seem to be.[424]

c. Consumer Characteristics Influencing Eating Patterns

Historically, most dietary interventions have focused on modifying the knowledge, attitudes, and behavior of individuals. Recently, strategies to achieve changes in eating patterns have expanded to include a greater consideration of the social and cultural characteristics of the individual's immediate or microenvironment, with respect to influences on eating behavior and health status.[419] Alteration of the microenvironment (in contrast to the larger environment) is more amenable to individual control.

Many theoretical models that seek to identify predictors of food behavior in individuals have not been validated. They lack the specificity required to quantify or qualify the various influences and how they relate to a specific individual.[420] In general, it is thought that two influences on food-related behavior are sociodemographic and psychosocial factors. These are reviewed briefly in the following discussion.

- Social and Demographic Influences on Eating Behavior

  Income. When personal income increases, the relative amount of money spent on food purchases decreases in comparison with other expenses.[425] This phenomenon, known as income-related elasticity of demand, is uncommon in the United States, presumably because the food supply is plentiful and relatively inexpensive.[420] Some foods have higher income elasticities than do others. Foods with high income elasticities include meats, fresh fruits, and vegetables.[426] Foods with low income elasticities include eggs, cereal products, fresh milk, and bread.[427] Purchase of dried beans, peas, rice, and eggs is inversely related to income. Two other items, low-fat milk and whole-grain bread usage, are positively related to income, perhaps reflecting the growing concern about health in the higher socioeconomic groups.[420] Nevertheless, the effects of income on food consumption patterns are still apparent. For example, data from the Nationwide Food Consumption Survey (NFCS) demonstrate that high-income households spend five times as much as low-income households on food eaten away from home.[427]

  Household Size. Larger households spend more money on food than smaller households, even at the same income level. However, the per-person food cost decreases as the number of family members increases. [428,429] The net effect on food purchasing behavior of larger household size is the same as a decrease in income elasticity. Thus, income spent on food eaten away from home is lower in larger households. In the 1977-78 NFCS, the foods most responsive to household size were fresh milk, dairy products, cereal products, bread, bakery products, sugar products, potatoes, fats and oils, and eggs, with some of these increasing and some decreasing as size of household increased. Surprisingly, meat usage remained remarkably high despite its high income elasticity, whereas the use of fresh vegetables, fresh fruits, and fruit juices decreased as household size increased.[429]

In regard to nutrient density, Windham et al.[430] reported that households with five or more members had a slightly but significantly lower fat intake than smaller households (difference of 2 grams per 1,000 calories). Households with three or more members had significantly higher carbohydrate intakes per 1,000 calories. In contrast, intakes of vitamins C and B_6 per 1,000 calories were significantly and inversely related to household size, as might be expected from the lower income elasticities for fresh fruits and vegetables in larger households.

Education. A number of studies have evaluated food behavior as a function of educational level for both males and females.[430-436] The most commonly reported association is a positive relationship between educational level of a female head-of-household and adequacy of dietary intake. Education-related elasticities of demand for fruits and milk are even higher than their income elasticities. Educational level has been positively correlated with nutrition knowledge,[431,437-439] but educational level and dietary behavior are not always positively related.[420] In more educated households, use of "convenience" foods is lower,[440,441] more meals are eaten together,[442] and more meals are eaten away from home.[440,442]

Gender. Gender differences in food and nutrient consumption in the United States are not striking. Women, because of their smaller size and lower energy needs, eat less than men, but the variety of foods they select is generally similar.[420,442] When nutrient densities are compared, women report consuming diets higher in vitamins A and C per 1,000 calories than men. Gender differences also have been noted in the Coronary Artery Risk Development in Young Adults Study (CARDIA), which is examining longitudinally the development of risk of coronary artery disease in 18- to 30-year-old black and white adults. Preliminary data from this study reveal that white women report dietary intakes that are more consistent with dietary recommendations for lowering high blood cholesterol than do males or black women.[444] In comparing male and female food usage using 1977-78 NFCS data, Cronin and colleagues[443] found no differences in 54 out of 65 food groups examined. In the 11 remaining groups, women reportedly ate more citrus fruit, yogurt, coffee, tea, and low-calorie beverages over the 3-day recording period, whereas men ate more whole milk, luncheon meats, meat, fish, and poultry sandwiches, desserts, sugar, and sweet spreads.[443] Women reportedly prefer fruits and vegetables more than men do, but within a specific culture such as that of the United States, the relationship between stated food preferences and food consumption is only low to moderate.[445,446]

Age. There are few longitudinal studies reporting the effect of aging on dietary intake. Most studies are cross-sectional, and thus they reflect not only the effect of age but also the effects of the dietary habits of different age cohorts. In the data reported, changes in nutrient intake patterns are generally consistent with the decreases in energy intake that occur with aging.[447-449] This is probably related both to a tendency of the elderly to be more sedentary and to a slowing of metabolism with aging. Data from NHANES I and II are currently being evaluated by age, gender, and ethnic background.[270] Preliminary results reveal that within ethnic groups, intakes of dairy products, meats, and low-calorie beverages are consistent throughout an age range within the population. The greatest differences appear in intakes of sugar-rich products, with those under 25 years of age consuming more of them.

Cultural, Ethnic, and Minority Considerations. Many important factors relating to health and nutrition in population subgroups have been described. Many of these differences are probably related to income rather than ethnicity, because income and ethnicity often vary simultaneously. Regional factors may also be operating.[450] Many of these differences are discussed in section VI, Ethnic, Cultural, and Minority Characteristics That Influence Diet and Health; others are considered here. As time in residence in a new country increases, the food habits of cultural subgroups gradually assume the characteristics of the dominant culture. Migrant studies support this observation, [342,420] including studies of first generation Chinese living in California, who reported reduced consumption of crab, oysters, prawns, and other foods popular in China, even though the foods were widely available in California as well.[451] Recent population-based surveys have reported differences in food group usage by age and race based on the NHANES II data.[270] The greatest black-white differences appear to be in the usage of dairy products. Blacks reported lower intakes of dairy products and increased intakes of sugar-rich products from age 12 onward.[270] This phenomenon may reflect, in part, dietary alterations resulting from lactose intolerance, which is common in blacks. Increased intake of other sugars is not easily explained on a physiologic basis, yet the NHANES II data suggest that blacks eat more sugar-rich products, jelly, jam, candy, and low-calorie beverages (diet soft drinks, coffee, tea, artificially sweetened drinks) than whites throughout life.[270]

Data from the Consumer Expenditure Survey of 1972-74 indicated that, compared with whites, blacks purchased more beef, pork, poultry, fish, and seafood but fewer cereal and bakery products, sugar-rich products, dairy products, and nonalcoholic beverages.[452] While some other studies confirm that blacks purchase more of these products than whites, evidence from NHANES II[270] is not as strong.

For other ethnic groups, there are few data on dietary intake of either a cross-sectional or longitudinal nature. Analyses of Hispanic NHANES data are now in progress. One study of Mexican-Americans reported that they consumed greater amounts of eggs, white bread, and tortillas but less dry cereal, pastry, wine, and beer than did a reference group of non-Mexican-Americans.[453] Both groups reportedly consumed the same amount of "convenience" foods, soft drinks, coffee, and tea.[453] These authors concluded that Mexican-Americans adopted eating patterns that were more like the stereotypical American patterns of several years ago, prior to the widespread interest in nutrition and health, than eating patterns reflecting either their culture of origin or their culture of residence

Employment Status of Female Spouse. The American family is changing; as more women work, the associations between female employment status and food-related behavior are of interest.[420] Most studies have reported that as the number of hours at work increases, and the number of hours spent doing housework decreases, people spend less time in meal preparation, [454-456] eat more meals away from home, and use more convenience foods.[440,442,457,458] However, not all studies have found this to be the case.[441]

Women's employment does not appear to be related to other factors such as food preparation style or the number of meals eaten together as a household. These factors are more strongly associated with the age of the children of the household.[420] The few studies that evaluate the relationship between quality of dietary intake from the standpoint of nutrients and employment status of the female head of the household [459,460] reveal no link between the two. There are few data on the effects on family nutritional status when households are headed by female single parents.[459,460]

- Psychosocial Determinants of Nutritional Status

Nutrition Knowledge. Nutrition knowledge influences food-related behavior, but the effect does not appear to be large.[431,435,437-439,461-465] Methodological inconsistencies may account

for the weakness of the positive correlations observed in most studies between nutrition knowledge and dietary intake.[420,466] Studies in which the food-related behavior of interest is specifically defined show the strongest associations with nutrition knowledge. Little information is available on the extent to which nutrition knowledge affects eating behavior among those who are deliberately attempting to alter their diet for health reasons.

Attitudes. Attitudes toward food and eating derive from a variety of different beliefs, feelings, and intentions, which are poorly documented but are thought to be important determinants of food habits. Food attitudes are also influenced by general health, health concerns, aesthetics, economics, convenience, creativity, adventuresomeness, sociability, prestige, familiarity, meal planning, and preparation styles.[420] Cultural and ethnic considerations also play an important role.[419] Correlations between food-related attitudes and eating behaviors are highest when the attitude and outcome measures are specifically defined.

Childhood Influences on Eating Behavior. Childhood influences on diet and eating behavior may carry over into adulthood. Biological, cultural, and psychological factors play a role in the food behavior of children.[467] During the first year of life, children learn what is edible and what is not. Once the edible foods have been identified, issues of taste preference arise. Some workers have suggested that there is an innate, but variable, preference for sweet tastes that is exhibited at birth and continues throughout life for most people.[468,469] It was long contended that infants left to their own choices would continue to have normal growth.[470,471] More recently, it has been recognized that this is not necessarily the case and that "hidden hungers" or the "biological wisdom of the body" do not ensure good nutritional status.[467]

In general, culture has the greatest impact on children's food choices and eating behavior.[468] Simply being exposed to a food increases children's liking for it.[470,471] Parents generally serve as the guides and gatekeepers to their children's food choices until children are able to obtain food on their own. Thus parents have a potent influence on children's food choices. However, siblings are more likely to have similar taste preferences with each other than even those between children and their parents. Preferences of mothers and fathers are also more highly correlated with each other than are those of parents with their children. Spousal correlations increase with years married.[468] Eating pattern correlations between parents and their children may be higher initially but are low by the time children are of college age.[472,473]

Much of the unexplained variability in food preferences within cultures is probably due to social encounters outside the family, especially between peers and respected adults.[468,474] Mass media and food advertisements in particular have capitalized on this method by selecting actors popular with children's audiences to endorse their products.

The nutrition education of children involves anticipatory guidelines for parents to help them avoid the "new faces of malnutrition in American children today," which consist of overnutrition, imbalances, and excesses of food intakes, sometimes coupled with poverty-related undernutrition such as vitamin-mineral deficiencies.[475]

Health System. Many parts of the health system influence eating patterns. The role of the Federal Government is exemplified by this report, along with efforts by the Surgeon General, the USDA, and many other groups. Many state and local departments of health supply nutrition services to groups such as children and pregnant women and to the general public (e.g., through publications and nutrition information sources). Physicians, registered dietitians, registered nurses, and other health professionals often provide nutritional guidance during illness as well as counseling designed to promote health maintenance.

D. Can Intervention Modify Dietary Behavior?

Historically, dietary interventions focused either on modifications of individual knowledge, attitudes, and behavior, or on changing governmental policies, regulations, or laws relating to food. More recently, interventions seeking to change individual behavior through environmental modification have been increasing.[476]

1. The Multiple Levels at Which Change is Possible: Individual, Network, Organizational, and Community

This section reviews selected interventions designed to change eating patterns. Such interventions have been implemented in many settings, such as schools, worksites, families, and fast food restaurants—and for many audiences, such as youth, adults, workers, restaurant patrons, and those with elevated blood cholesterol. The results of these programs can be categorized according to the level of the target: individuals, social networks, organizations, and communities, states, or societies. A framework for reviewing blood cholesterol lowering interventions at these target levels is presented in figure 8, page 76.

Primary prevention programs are often planned to encourage individual behavior change in order to reduce risk for a particular disease or health problem. For example, a blood cholesterol-lowering program to reduce CHD risk might encourage people to eat more fruits, grains, low-fat dairy products, and vegetables, and to consume only moderate portions of lean meat, poultry (without skin), or fish in place of high-fat meats, poultry fried or with skin, or fried fish. However, some have argued that to be truly supportive of individual changes, changes at the social network, organizational, and community levels must also occur.[477,478] This approach views the individual as part of a larger system made up of levels of social organization that are increasingly complex. Each level, or part of the system, affects and is affected by the individual's health behavior. Brody and Sobel[479] argue that "disease most often involves multiple levels disrupting the person and social group, and therefore requires multiple interventions directed at different levels." This supports the idea that family, friends, and the community at large can play important roles in modeling and reinforcing changes in the individual's health behavior.

Targets of change at the *individual* level include: youth, the elderly, women, men, ethnic and racial minorities, the poor, and those at high risk. Interventions at this target level include interpersonal strategies such as classes or individual dietary counseling including telephone-mediated approaches; [480] minimal contact strategies such as self-help printed materials, home videotapes, or special television programming; and technologies such as computer-based assessment and feedback.[481,482] *Social-network*-level interventions target families and larger social networks such as circles of friends as a means to influence dietary behavior. This level of intervention uses similar strategies to those of individual-level interventions (e.g., interpersonal, media, and technologies), emphasizing that information be disseminated by key persons such as opinion leaders.[483,484] *Organization*-level interventions seek to make changes in organizations such as worksites, restaurants or restaurant associations, food boards, and physicians' offices. Some examples of strategies for change at this level are to increase the amount of health-promoting information in the organizational environment; to provide programs to initiate and support dietary change (such as offering classes and stocking vending machines with low-SFA and -cholesterol foods); and to promote organizational policies on types of foods advertised or sold.[485,486] *Community*-level interventions are intended to affect the entire community (e.g., entire populations of individuals, organizations, or networks). Strategies include using the mass media to persuade and educate, implementing health-promoting policies and regulations, and changing the activities and programs of health agencies (e.g., the addition of blood cholesterol screening programs and changes in the curriculum requirements of institutions of

Figure 8

A Multilayered View of Cholesterol-Lowering Strategies: Levels of Intervention and Corresponding Targets of Change

INDIVIDUAL-LEVEL TARGETS

Men/Women
Youth
Elderly
Minorities
Low income

NETWORK-LEVEL TARGETS

Extended social network
Peer groups
Families

ORGANIZATION-LEVEL TARGETS

Worksites
Restaurants
Grocery stores
Schools
Cafeterias

COMMUNITY-LEVEL TARGETS

Media
Social norms
Public opinion
Local legislation
Food production
Institutions of higher education

higher education). Many examples of such community programs exist in the United States and abroad.[347,487-490]

This section reviews selected research results from cholesterol-lowering and dietary change programs at each of the four levels. These studies have been selected primarily because they: (1) investigate a diet change/cholesterol-lowering intervention that is congruent with the NCEP goals for cholesterol lowering,[491] (2) are related to primary prevention of cardiovascular disease, (3) exemplify one of the four levels of intervention, and/or (4) include a rigorous evaluation. Also included are relevant interventions in other health areas that illustrate important and successful models for behavior change and that have not been conducted for blood cholesterol reduction. Not included are weight loss and exercise programs, which, although they may have a substantial influence on blood cholesterol and CHD risk, are beyond the scope of this review.

2. Individual-Level Interventions

Various strategies may encourage individuals to change their diets through individual-level interventions. Typical are interpersonal strategies such as lectures, counseling, or groups and classes, supplemented with written materials. Intensive individual dietary counseling, group programs, or adjunctive counseling in community studies also can have favorable effects on both eating patterns and blood cholesterol level.[343, 344, 492-495] Combinations of mass media and individual dietitian counseling have also proven to be effective.[346, 496] In addition, minimal contact programs have been used increasingly to inform and to teach dietary change skills.

a. Individual and Group Face-to-Face Interventions

Efforts aimed at increasing adherence to dietary modifications for health purposes, especially efforts to lower blood cholesterol, require motivation and encouragement from all of those involved. Successful methods for one-to-one health care encounters and in group situations have been described. Meichenbaum and Turk,[497] for example, suggest several steps that ensure adherence to modified eating behaviors: "anticipate nonadherence; consider the prescribed self-care regimen from the patient's perspective; foster collaborative relationships based on negotiation; be patient-oriented; customize treatment; enlist family support; provide a system of continuity and accessibility; make use of other health care providers and personnel as well as community resources; repeat everything and don't give up."

In the past two decades, nutrition education and dietary counseling for blood cholesterol lowering have become popular. Several examples of such counseling strategies are available.[483,498,499] In several lipid-lowering investigations such as the North Karelia study, MRFIT, and the LRC-CPPT, individual counseling has been used effectively (in North Karelia, individual counseling supplemented group and mass media efforts).[323,343,344,346] Snetselaar[500] has reviewed methods that have proven effective in blood cholesterol lowering with individuals. These methods can be used for groups as well.

Johnson and Johnson[501] identified 673 studies of nutrition education programs conducted since 1910, and analyzed 303 programs that had been quantitatively evaluated. The analysis was restricted to programs that had measurements of the participants' attitudes, knowledge, and behavior on diet, and either a pre- or post-change score or a score comparing treatment with control groups. Some of the major programs included in the analysis are the Special Supplemental Food Program for Women, Infants, and Children (WIC), Expanded Food and Nutrition Education Program (EFNEP), and the National Dairy Council's Food...Your Choice.* All of the programs used individual-level change strategies, with lectures, written materials, and directed small group activities ranking at the top.

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* It should be noted that the dietary objectives of these studies varied. They are included here because they were dietary behavior change programs, not because of the actual changes they advocated.

The findings of Johnson and Johnson[501] suggest that individual-level dietary change programs are successful about half of the time. While very few programs produced negative results (i.e., participants did not regress in their knowledge or behaviors), many programs had no measurable effect after the intervention. Knowledge and attitude were significantly changed in about half of the programs. Somewhat more than half of the studies showed significant differences in behavior change. Of the total of 4,108 findings from the 303 studies, 67 percent focused on consumption of nutritious foods. The changes observed were reported for all age levels, for both males and females, and for all socioeconomic levels regardless of the type of individual-level instructional procedures used. The results were especially strong for positive changes in the consumption of fluid milk, of dairy products as a whole, of meat and fruits, and of protein, fat, calcium, thiamin, riboflavin, and niacin.[501] This analysis did not review the effects of the dietary changes on blood cholesterol or other physiological indices related to diet.

In another review of nutrition interventions for reducing chronic disease risk factors, Glanz[502] categorized the individual-level interventions into two types: nutrition education and nutrition counseling. Nutrition education is defined as the process of imparting knowledge that is aimed at the general improvement of nutritional status through elimination of unsatisfactory dietary practices, promotion of adequate food habits and greater food hygiene, and more efficient use of food resources.[502,503] Nutrition counseling is described as an individualized process by which a person is helped to acquire the ability to manage nutritional care, to gain nutritional knowledge, and to improve decisionmaking and behavior change skills.[502] In practice, the distinction is primarily that of the size of the audience; in counseling the audience is one individual, whereas in nutrition education the audience is most likely to be a group or population. Moreover, counseling tends to be more focused with respect to goals (e.g., reduce SFA intake to lower blood cholesterol) and usually takes place when an individual is motivated to change. Nutrition education, on the other hand, does not necessarily assume that motivation exists or that goals for change have been identified.

Intervention strategies have been classified as instructional, motivational, or behavioral. Instructional approaches are concerned primarily with imparting information about disease processes or eating patterns. Motivational strategies focus on verbal persuasion to change attitudes and increase motivation to change; they do not usually include use of incentives. Behavioral approaches to change identify the antecedents and consequences of behavior, determine barriers to change, and arrange behavioral contingencies to facilitate change.

Reports of nutrition education programs are rarely detailed enough to determine the ways in which these three strategies differ. A few comparative studies seem to indicate that behavioral strategies may be more effective than other approaches in achieving dietary behavior change.[502,504] However, this has not been rigorously evaluated in terms of physiological endpoints, such as changes in blood cholesterol level. The theoretical basis that governs the choice of each of these strategies is rarely reported, especially when instructional and motivational strategies are used. Studies of behavioral strategies more commonly report the theoretical base, usually social learning theory,[505] which postulates that interactions among individual and environmental factors augment the likelihood of behavior. Specific details on the behavioral strategies studied are beyond the scope of this review.

Glanz[502] reviewed 10 studies on lowering dietary fats and found that 7 produced changes in blood cholesterol via nutrition counseling or education. The context of these interventions ranged from clinical trials and community studies to worksite investigations. Approximately one-third of the study results that related to low-fat, low-SFA diets were from community studies, each of which carried out nutrition education and counseling as part of a comprehensive dietary change strategy. The other two-thirds involved individual strategies. No single strategy was consistently the most successful or cost-effective. Many of the studies aimed at changing multiple risk factors. Thus, the possible independent contributions of smoking cessation or changes in physical activity or nutrition education are unknown. Most studies failed to demonstrate differences in educational strategies, but the intervention groups showed improvements compared with controls.

Glanz[502] has also reviewed dietary interventions for a number of outcomes in addition to lowered blood cholesterol, such as treatment or control of hypertension, reducing the risk of cancer, and weight loss. This study recommends that nutrition education programs be tailored to individuals' needs, include social support, supply skills and information in nutrition education materials, provide adequate patient-provider communication, and pay attention to patient followup, monitoring, and feedback. A recent review by Disbrow,[506] focusing on the cost-benefit relationship of nutrition services for the prevention of CHD, has summarized many individual and group counseling programs and found that such approaches are both promising and cost-effective.

b. Individual Interventions Involving Minimal Contact With Providers or Facilitators

Several promising individual-based strategies to assist dietary change have emerged in recent years. One is the use of materials that require minimal contact with providers. Such programs are behavior oriented, require minimal therapist time, are disseminated through a variety of channels of communication, and rely to a great extent on self-administration by the individual.[507] Features include self-help printed kits and booklets; electronic media programs (radio, television, or home video); and new technologies, such as computer software, electronic mail, and other computer-based programs.

For example, researchers working with the Stanford Five City Project (FCP) developed a skills-oriented dietary change "kit," consisting of four pages that outlined steps for changing eating behavior. In a pilot study of the effectiveness of the kit, FCP workers compared a traditional nutrition information booklet with the skills-oriented, self-help kit. Participants in the study were recruited through the mail and were randomly assigned to receive the booklet, the kit, or no materials for 6 months. Results indicated that use of the kit significantly reduced reported dietary intake of cholesterol and increased nutrition knowledge at 6 weeks and 29 weeks after completion.[508]

A second minimal-contact program developed in the context of the Stanford FCP was a multiple-risk reduction newsletter-based project titled "Healthy Living." More than 6,000 self-selected individuals signed up for and received the newsletter over a period of one and a half years. Most participants were female, over 35 years of age, and interested in health. The newsletter contained recent health information, tips for changing behavior, stories of people in the comunity who were making health-related changes, and news about health promotion programs, materials, and presentations available locally. The program was evaluated by surveying a random sample of participants before and after the program. Results showed no consistent behavior changes but increases in perceived ability to choose "healthful foods."[509] It is not known whether this perceived ability may influence future behavior.

As a third example, computer-based dietary assessment and feedback was a key element in a collaborative effort between Stanford researchers and private business aimed at reducing cardiovascular disease risk in the workplace.[510] At the beginning of the program, participants viewed a series of health videotapes and were counseled by a specially trained nurse. The information provided after the initial and monthly computer-based assessments and feedback was enhanced by attractive materials with tips for changing eating behavior. This computer-aided risk reduction effort led to statistically significant changes in blood

cholesterol (and smoking) in these workplaces when compared with a randomly assigned control workplace within the same corporation.[510] This same group of researchers carried out a second cholesterol-lowering intervention in a worksite using a brief questionnaire of the frequency of consumption of selected foods, computer-generated individualized feedback, brief counseling at the time of assessment, printed materials on dietary goals, and dietitian-initiated phone contacts on progress. This minimal-contact, individualized treatment resulted in a significant reduction in total cholesterol in the treatment group (n=114) from 238 ± 25 mg/dL (6.15 ± 0.65 mmol/L) to 223 ± 33 mg/dL (5.77 ± 0.85 mmol/L)($p<0.002$). One-third of participants in the treatment group had a 20 mg/dL or more decrease.[480]

Finally, incentive-based programs, such as lotteries or contests, have been shown to be a cost-effective means of helping individuals quit smoking and lose weight.[511] Incentives have a potential role in reinforcing and maintaining behavior changes.

c. Nutrition Education and Dietary Change Programs for Groups of Children and Adolescents

Individual-level interventions have also been designed for children and adolescents. These programs often are found in school settings and use educational and skill-based strategies to change the dietary habits of young people.

School-based programs for children. In a review of school-based nutrition education programs, Saylor and colleagues[512] concluded that most interventions are informational rather than skill-based, are not theoretically derived, use self-report change measures, and often do not have control groups and long-term followup.

There are, however, some exceptions. Coates and colleagues[513] reported that a 12-class program for fourth- and fifth-grade students, which also targeted parents, produced changes in the children's choice of school lunches and in the parents' self-reported eating behavior. While exercise was also targeted for change, the results were less substantial. The curriculum, delivered by university undergraduates, included techniques such as modeling, behavioral rehearsal, goal specification, feedback, and reinforcement for behavior change. Perhaps most important, the study included observation of the students' behavior to assess the outcome of the program.

Researchers at the University of California at Los Angeles produced "Know Your Body," a chronic disease risk reduction program for elementary students. Youth from 18 schools were assigned to one of four comparison groups: curriculum and health screening, curriculum alone, screening alone, and control. One of the nine curriculum modules focused on nutrition. Students in all three of the intervention groups scored more points on all six measures of knowledge of caloric value, nutrients, and basic concepts of blood cholesterol than did controls.[514] Eating behavior was not observed, but self-reported behavior was measured. The treatment groups and controls did not differ in consumption of dairy products and foods high in SFA and cholesterol. However, "high-risk" students (49 percent of the screened population) in the screening-and-curriculum group did report such dietary changes.

The first application of the "Know Your Body" program was with New York elementary school children in 22 schools from either a lower-income district in the Bronx or an upper-income suburb in Westchester. Schools were randomly assigned to intervention or control groups. Beginning in the fourth grade and continuing to the eighth or ninth grade, students were taught the "Know Your Body" curriculum. The principal findings from this longitudinal study were that the program was associated with favorable changes in blood cholesterol level and the rate of initiation of cigarette smoking. No effect was seen on body mass, physical fitness, or blood pressure. The researchers report that the findings may be generalized to similar populations of school children elsewhere.[515,516]

Application of the "Know Your Body" program with black elementary school students in Washington, D.C., indicated that after 1 to 4 years of "Know Your Body," changes in the desired direction occurred for blood pressure and blood cholesterol level. The differences between the intervention and control groups were consistently statistically significant only for diastolic blood pressure.[517]

The Minnesota Healthy Heart curriculum is a 5-week, 15-session school curriculum taught by third-grade classroom teachers. The program targets changes in environmental, personality, and behavioral factors that are likely to influence children's eating patterns. The study results indicated changes in the desired direction for 5 of 12 targeted foods.[518]

These three examples, particularly the two resulting in behavior change,[513,518] highlight the potential for changing dietary behavior of young children. These successful programs (1) integrate behavior change theory into the development and implementation of curricula, (2) use behavioral rehearsal and role playing as part of the intervention, (3) focus on specific foods rather than general information, (4) have a component that includes parents and children working together to make changes, and (5) concentrate on skills for behavior change combined with factual information.

School-based programs for adolescents. Working with seventh graders in Finland,[519] the North Karelia Youth Project compared a community-based intervention with a combined school- and community-based intervention. The goal of the ten 45-minute sessions was to reduce the five risk factors for cardiovascular disease. The researchers noted a significant decrease in fat intake (measured by amount of fat in milk) among the intensive school and community intervention groups compared with school and community groups that received no special instruction.

A dietary intervention school program for older adolescents based on an earlier program delivered to elementary students and parents was designed by Coates and colleagues.[513,520] The new program with black high school students focused on food behaviors particularly salient to adolescents (snacking), used behavior principles to develop the intervention, conducted an in-school media campaign, and concentrated on reducing intakes of salty, high-fat, high-SFA, and high-sugar snacks. The study compared classroom instruction with and without a parental involvement component. Initial changes in the desired direction for snack foods were not maintained at followup except for the group with the parent involvement component; these students reported eating fewer target snack foods. Study investigators also reported that participation on a sports team predicted desirable snack consumption, while beer drinking and smoking were associated with undesirable snacks.

In a study by King and colleagues,[511] a 15-session curriculum, taught by specially trained instructors, focused on snack foods, utilized social learning theory, incorporated behavioral measures, and conducted a long-term followup. At the end of the program there were significant changes in knowledge, reported availability of healthful food at home, and self-reported behavior change. At 1-year followup, knowledge was still greater among students who had participated in the program.

In Texas, two of four elementary schools were assigned to a diet and exercise intervention group and two to a measurement-only group. The intervention group included third and fourth graders whose teachers received two in-service programs twice each year. The school curriculum was modified to include two 4-week healthful eating modules and a 6-week physical activity module. The students were taught to recognize foods by their sodium and fat content and practiced making healthful food choices at fast food restaurants and at parties. The school lunch menu was modified to be lower in fat and sodium. The results indicated that, in the intervention groups, self-reported salt use declined in comparison to controls; however, there was no statistical difference in reported lower fat food selections. The authors note that changes in one intervention group were offset by improvement in control groups due to secular trend, contamination, or social desirability.[521]

Another study of dietary modification of adolescents was conducted by Perry and colleagues[522] in Minnesota. This pilot study for the Minnesota Heart Health Program was unique in its curriculum design and findings. Approximately half of the 10-session curriculum for the participating tenth graders was presented by classroom peers. Knowledge, food choice, and self-reported diet and salt intake were measured. While girls in general performed better on all measures, boys and girls made significant changes in knowledge and salt intake scores.

These studies illustrate the utility of theory, the need for rigorous research design and evaluation, the necessity for behavioral and physical outcomes, and the benefits of long-term followup. The Minnesota study points out the need to tailor messages to audience subgroups for maximal effectiveness.[522] Except for the Coates study,[520] minorities were not analyzed separately.

3. Network-Level Interventions

Network-level interventions attempt to modify the quantity of food-related communications within a network, defined as "the web of ties that surrounds an individual."[523] Despite its potential for health education, this type of intervention is the least studied of the four levels. Such programs present difficulties such as achieving and sustaining adequate access to participants.

Networks encompass family members, friends, members of a church or club, or any other formal or informal contacts one has on a regular basis. In a research context, networks are studied according to the quality of interactions, and their frequency, intensity, durability over time, and strength.[523]

a. Family Networks

While there are numerous studies of the effects of social networks on health status, only a handful of programs have intervened at this level. Families are the most common target for such interventions (see the successful results of youth programs that have incorporated family members, above). Intervention strategies used with families have included interpersonal communication such as group meetings and discussions and print media such as handouts and direct mail.

Minnesota researchers compared a home-based eating patterns program for third-grade children to a school-based approach. The "Home Team" program, a 5-week correspondence course with the children and their parents, mailed weekly packets containing behavior tip sheets and worksheets to the children's homes. The packet described 2 to 3 hours of activities concerning eating pattern changes. Finally, a grand prize was offered as an incentive for participation, as evidenced by participation points. The results showed that the self-reported food selections, the 24-hour dietary recall data for fat, SFA, and complex carbohydrate consumption, and the food-shelf inventory for encouraged foods were all more positive for children and homes involved in the "Home Team." At a 1-year post-test, most of the changes were no longer significant. In general, associations were statistically stronger when the student and not the school was the unit considered in the analysis.[524,525]

Another study in California evaluated the effectiveness of a family-based cardiovascular disease risk reduction program in two ethnic groups.[526] The program reached 206 low- to middle-income Mexican-American and non-Hispanic white (Anglo-American) families with fifth or sixth graders. Half of the families were randomized to a 1-year intervention to decrease intake of foods high in fat, SFA, and sodium and to increase regular physical activity. The intervention sessions, taught by two graduate students, included 12 intensive, weekly sessions over a 3-month period and 6 maintenance sessions taught monthly or bimonthly for the subsequent 9 months. Followup continued for 24 months. The results indicated an increase in knowledge of the skills needed to change diet and exercise habits in both Mexican-Americans and Anglo-Americans, compared with the control groups. The intervention groups reported improved diets in the food questionnaire. There were no significant differences in physical activity. A significant decrease was shown in LDL-cholesterol levels for Anglo versus control participants. There were significant differences in blood pressure levels, which were 2.2 to 3.4 mm Hg lower in both Anglo- and Mexican-American intervention groups than in the control groups.

b. Peer Networks

Researchers have suggested several models of communication flow for understanding peer network influences. For example, an intervention could influence the target audience directly or by reaching the target audience indirectly through opinion leaders and other people influential in the network.

Using the indirect approach to promote diffusion of health information, the North Karelia project trained lay health opinion leaders to promote the dietary and other messages central to the prevention of cardiovascular disease.[527] These interpersonal messages reinforced similar messages broadcast in the media, presented at classes, and provided through events in the community. Thus, several levels of intervention provided stimuli for change to the networks.

A second strategy is to actually develop social networks. The Stanford Three-Community Study (TCS) showed that opinion leadership emerged from high-risk participants who were exposed to both media-based and interpersonal intervention.[528] As the magnitude of input increased (from mass media only, to mass media plus screening plus change agent contact), so did the number of contacts and the number of health conversations. This study illustrated how the media alone and the media supplemented with interpersonal contact affected the diffusion of health information. Similarly, in a recent study, groups of women at risk for cancer used self-help networks to achieve reduced dietary fat intake over a period of 2 years.[529]

Promoting changes in communication behavior within social networks may be another important avenue for dietary change. While the effect of the content of network relationship on health outcomes has not been directly studied, spouse and peer support have been shown to be important elements in health behavior change and maintenance.[526,530,531] Both the number of supportive interactions and the kinds of supportive interactions may be important. Changes in network interactions about diet and health can potentially influence the short- and long-term outcomes of programs in organizations and in whole communities.

4. Organization-Level Interventions

As intervention targets, institutions and organizations such as worksites, schools, primary health care settings, supermarkets, and other retail food outlets offer many potential benefits. The two most studied settings for organization-level interventions are the worksites and point-of-purchase settings such as cafeterias, restaurants, and grocery stores. There are also several studies of schools as settings for organization-level change. Using organizations as channels for delivering health programs, health educators can reach target audiences; tailor interventions to specific contexts; multiply efforts via social support, instructional expertise, and facilities; influence organizational structure in ways that will support individual-level behavior change; and persuade organizations to become healthful models for other community institutions.

McGandy et al.[532] changed the foods available in a school food service and found lowered blood cholesterol levels in students. Ellison et al.[533] have also attempted cardiovascular disease modification by a change in the school environment, especially by reducing the fat and sodium content of food made available through the food service in two boarding high schools. By implementing the program in each of the schools in alternate school years, researchers were able to compare students at a school in which there were food service changes and students in a school without such changes. These students were not counseled to change their eating practices since the intent was to study environmental changes, but they were asked to complete periodic 24-hour food diaries. The study demonstrated that, at the end of the academic year, the change in food service was associated with an estimated 15-20 percent decrease in sodium intake. In addition, preliminary results indicate that total fat decreased about 9 percent from 35.4 percent of calories at baseline to 32.1 percent and that SFA decreased 21.8 percent from 12.4 to 9.7 percent. The latter study showed the effects of an environmental program alone and cannot predict the influence of an additional education component.[533]

Typically, interventions delivered through organizations target and measure individual change. Recently, however, some research has targeted and measured both organization- and individual-level change.[521,531] In one weight-loss program, for example, organizational goal setting, public displays of group achievements, and use of the organization's newsletter, as well as intergroup competition were used to enhance generalization and maintenance of lifestyle change. The individuals demonstrated weight loss at a 6-month followup, and the workplace found the program feasible to maintain.[531]

Organizations not only can serve as channels for reaching individuals, but can also be targets of change. In terms of CHD risk and food, these organization-level interventions attempt to change conditions or policies within an organization in order to promote diets likely to lower blood cholesterol levels. They are characterized by strategies to increase the availability of appropriate food choices, improve access to these foods, increase the appeal of low-fat, low-SFA, and low-cholesterol foods, increase the information about these foods, and create incentives to eat healthful foods.[478]

a. Worksites as Targets of Change

In a review of nutrition programs at the worksite, Glanz and Seewald-Klein[534] found that most worksite health promotion programs offering nutrition education have been developed since 1980. The most commonly used educational method is printed brochures or newsletters. These authors report that the Johnson and Johnson Company's Live for Life program, based in part on an eight-session nutrition education course, is reinforced with policy changes that provide scales in restrooms and food choices that are low in calories and fat in vending machines and the cafeteria. An evaluation of this program concluded that there was a significant decrease in the number of overweight employees at program sites. In addition, Southern New England Bell Telephone Company's 6-week nutrition course, plus short courses and point-of-purchase information at company cafeterias, resulted in measurable reductions in weight, blood triglycerides, and cholesterol.[534]

Evaluation of two other worksite nutrition programs provide some insight into components that are linked to successful behavior change. In a worksite screening and education program for employees of the Social Security Administration, the treatment group received individual counseling, and spouses were invited to an informational meeting. Although the program resulted in a reduction of blood cholesterol levels by 12-17 percent compared with 4 percent for the control group, these results disappeared after the completion of the program. The Heart Disease Prevention Project of the World Health Organization, conducted in places of work, found that cholesterol reduction was greatest when participants received interpersonal communication about diet rather than print communication.[535]

The New York Telephone Company's short nutrition course was designed for high-risk individuals with total cholesterol levels over 240 mg/dL (6.21 mmol/L). The treatment consisted of an eight-session program with monthly behavior maintenance meetings for the following 6 months. Cholesterol declined a little more in the treatment group (8.8 percent) than in the control group (6.4 percent).[504]

These studies suggest that worksite-based classes and individual counseling can be effective, and that individual dietary change can be supported and enhanced by changes in the environment such as providing low-cholesterol entrees in the cafeteria. Recommendations for enhancing the effectiveness of worksite programs have been published,[536] and the American Heart Association has developed a detailed worksite program entitled Heart at Work.[537]

b. Point-of-Purchase Interventions

Point-of-purchase interventions are those conducted at sites where people purchase food either to prepare or to eat directly.[538] These programs aim to influence the food purchases people make; however, learning, deciding, choosing, and consuming may also be targeted outcomes at point-of-purchase sites.[539] Restaurants, grocery stores, cafeterias, "mini" markets, and even vending machines are all examples of potential sites for point-of-purchase interventions.[540] Generally, desired institution-level outcomes are increased availability, accessibility, and purchase of healthful foods, along with the improvement of the institutional environment through increased information and reinforcement of healthful actions. One would typically work toward these general goals by increasing the number of advertisements for healthful food within the store, labeling the healthful foods, organizing food tastings for targeted foods, and distributing informational brochures.[541-544] Little has been done to test the effectiveness of changing the in-store environment (e.g., optimal positioning of healthful food on shelves or packaging of foods to encourage consumers to eat smaller amounts, such as providing 4-ounce packages of meat to yield 3-ounce cooked portions), but some observations have been made.[545] Researchers at the Minnesota Heart Health Program have been successful in collaborating with local beef and pork producers to provide consumers with information about lean cuts of red meat.[476] Training meat department personnel and taste-testing recipes are all part of the educational effort.

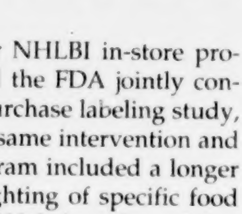
While not yet examining all the potential means of influencing people in food outlets, studies of point-of-purchase interventions have already yielded some important results.[546] Although the impact of these interventions on individual eating patterns and health outcomes is unclear, they have demonstrated that organization-level change is possible.

Grocery Store Programs. Programs in grocery stores use nutritional information displayed at various points in the store as a means of increasing the knowledge of consumers and promoting a change in food purchasing behavior.[547] In a national survey of 83 grocery store chains, Pennington and colleagues[548] found that only 30 (36 percent) had some type of in-store food health labeling program. Moreover, there was a lack of consistency across programs in the use of terminology, labeling format, and color coding of calorie and nutrient labels (such as "low calorie"), indicating a potential for consumer confusion.

A number of studies of grocery store intervention programs have placed particular emphasis on purchase behavior as an evaluation criterion. The studies have measured changes in the actual sales of entire food categories or specific brands targeted for health-related nutrient characteristics. A cooperative program between NHLBI and Giant Food Inc. in the Washington, D.C., area—"Food for Health"—used special brochures and sectional shelf labeling over a 1-year period. Evaluation results indicated a significant increase in knowledge at the intervention sites compared with a control group of stores in Baltimore. The campaign also demonstrated the feasibility of point-of-purchase information programs and generated a high demand for materials. Sales data, however, did not indicate a shift in food purchases for intervention versus control stores.[549,550]

In another early attempt to influence food behavior through grocery store interventions, Jeffery and colleagues[551] carried out an experimental study in eight Minnesota stores over 6 months. They monitored weekly sales of 25 items and administered pre- and post-test questionnaires to shoppers on dietary knowledge. Although shoppers' knowledge increased in both experimental and control stores, there were no differences in food purchasing behavior.

Building on the earlier NHLBI in-store program, Giant Food Inc. and the FDA jointly conducted a 2-year point-of-purchase labeling study, "Special Diet Alert," in the same intervention and control markets. This program included a longer test period and the highlighting of specific food brands by both special shelf labels and an alphabetized booklet containing the names of all participating brands. The average market share of flagged



products increased by a statistically significant 8 percent in intervention versus control stores for 8 of the 16 product categories.[552] The results of this study were interpreted in the context of a review of seven in-store purchase behavior studies reported in the literature (two with positive and five with negative results). This review led to the conclusion that positive outcome was related to some combination of a longer program duration, simpler nutrition messages, brand-specific labeling, and experimental or statistical control of environmental variables in the supermarket.

The FDA-Giant study was enlarged to include more brands and food categories and was placed for an additional 2 years in stores in Baltimore, the control market for the original study. Original test market stores in Washington also carried the expanded program. A nominal price was charged for the in-store booklet. Statistically significant market share gains occurred for labeled foods in Baltimore stores in half of the product categories that had been part of the initial program. Furthermore, in 25 newly added product categories, flagged products had significant market share gains in 10 categories as opposed to share declines in only 4.[553] In-store booklet sales were not strongly related to usual shopper demographics, but were concentrated among shoppers with a household member on a special diet. The booklet purchase rate averaged 9 percent of households patronizing the test stores; this equaled the average U.S. household redemption rate for direct mail food coupons during the same period, according to national clearinghouse statistics. The use of food coupons is considered an effective means of persuading consumers to try products, and the nutrition booklet purchases compared quite favorably to coupon redemptions. Across stores, average booklet sales were moderately and positively correlated with the magnitude of gains in the market share of labeled products.[554]

The FDA recently completed a third in-store labeling program of similar design to the Giant Food study in cooperation with the Stop and Shop food chain in the New England area. Preliminary results in 60 participating food categories essentially replicated the findings of the earlier studies, with statistically significant gains in market share registered for labeled products in half of the categories. The shifts in purchase behavior occurred in markets that were smaller, as well as sociodemographically and ethnically more diverse, than the Washington and Baltimore markets.[555]

The Minnesota Heart Health Program initiated a community "Eating Pattern" task force that developed the "Shop Smart for Your Heart" program. This consisted of an integrated system of shelf labeling, posters, shopping bag stuffers, a preprinted grocery list, and a brochure all emphasizing low-cholesterol, low-fat, low-SFA foods. Telephone surveys of randomly selected participants made 2 to 4 months after the start of the program showed that in the two intervention cities, 25 and 47 percent of the respondents, respectively, reported that labels influenced their food purchasing decisions.[556]

The Pawtucket Heart Health Program introduced, in 1983, the "Four Heart" Grocery Program, which used shelf labels, product-specific cards, posters, and brochures to inform consumers about foods low in fat, cholesterol, or sodium. The program was evaluated using consumer exit interviews and food sales. The result showed that in-store programs increased consumer awareness and influenced their food choices.[557]

Knowledge of the outcomes that can be expected from point-of-purchase programs is incomplete; there is ample evidence that consumers are aware of the programs, are satisfied with their content, and report that the programs influence their food purchases. The growing body of sales evidence confirms that shifts in purchase behavior do in fact occur in many food categories, but there is no evidence to date on whether the new purchase habits are sustained over time.

Restaurants and Cafeterias. Point-of-purchase programs also have been implemented in businesses that sell prepared foods. These include restaurants, cafeterias, and convenience food outlets. These programs also rely on print media to educate and persuade consumers to buy particular foods. One study used a single poster advertising the health benefits of the low-fat entree, which resulted in a 15 percent increase in purchases of that entree.[558] Wagner and Winett[559] replicated these findings by successfully using signs and posters to cue healthful food choices in a fast food restaurant. They found that the cues for salads increased the sales of salads; sales of higher fat foods such as hamburgers and french fries did not decrease. By contrast, a study of the impact of nutrition information placed in vending machines concluded that availability, rather than attractive nutrition information, was the best predictor of whether a consumer would purchase a food item that was lower in calories, sodium, and fat than other choices.[560]

In a study of a restaurant menu labeling program in four family-style restaurants in four communities (two in the Stanford Five City education communities), Albright and colleagues[561] found that sales of targeted entrees increased significantly in two communities and stayed the same in two other communities. Neither food sales data nor exit surveys of patrons explained the differences in impact.

Cafeterias serve millions of students and employees in the private and public sector. A game developed to promote healthful food choices in a large institutional cafeteria was associated with reduced purchases of desserts and total calories per customer and increased sales of skim milk compared with controls.[486] Another cafeteria-based program, using labels to emphasize the calorie content of foods, found reduced purchases of high-calorie side dishes but not entrees so that the overall calorie content of meals purchased was minimally affected.[562] At a university dormitory cafeteria, a food labeling program was developed that consisted of food labels with information about the amount of fat, cholesterol, and calories for each item. Compared with the baseline data, selections of meals lower in fats, cholesterol, and calories increased from 31 to 47 percent depending on the meal and intervention phase; selections of such meals remained higher than baseline levels 5 weeks after the intervention stopped.[542] In another cafeteria program, Cincirpini,[563] over the period of an academic year, assessed the effects on food purchases of various methods, i.e., calorie listing, labeling of target items, and token rebates (incentives) for labeled items. The tokens produced the most uniform changes toward low-fat and low-calorie food choices.

Point-of-purchase programs in grocery stores, restaurants, and cafeterias are feasible and in most cases welcomed by store managers.[556,564] Consumer satisfaction and awareness are typically increased after intervention.[476,556] Consumers report that labels and other informational cues influence their choices, yet sales data do not consistently confirm these reports. This may be due, in part, to other factors that also influence shifts in sales, such as price and seasonal variation. In addition, many studies lack controls, thereby rendering interpretation difficult.

There has been little comparison of different types of approaches to promote healthful foods. The usual messages are informational and emphasize health values. In fact, other factors such as price, agricultural season, culture, and geography may influence food purchasing behavior. Colby and colleagues[565] compared different influences for a low-fat entree in a family restaurant: health, price, taste, and no special influence. Sales data showed that consumers were more likely to purchase the low-fat entree promoted as a low-priced daily special.

Overall, labeling programs in grocery stores and restaurants have yielded mixed results. In the literature reviewed, consumer knowledge seems to have increased in all of the studies that measured knowledge change. Evidence of behavior change is less consistent, although each of the more recent supermarket studies has demonstrated shifts toward more healthful food purchases in about half of the food categories studied. Successful grocery store programs have combined the following elements: a focus on specific foods; an emphasis on a few diet/health issues; and a program duration of more than 1 year. In addition, they have used a rigorous experimental design for program evaluation. Considering the food distribution channels as a whole, however, results concerning the amount of labeled products are inconsistent. Some studies document changes while others do not. Increases in purchases of identified foods have been reported in some instances without a corresponding decrease in the amount of purchase of foods that are high in fat, SFA, cholesterol, and calories.

c. Schools as Targets for Change

Many workers have considered schools as settings for influencing individual behavior; schools themselves can also be targets of change.[566] Redefining the cholesterol, fat, and sodium levels in school lunches is a policy change with far-reaching implications.[567] A study of school lunches in Texas revealed that local school lunches had 30 percent more fat and 100 percent more sodium than recommended by the study standard.[521] Thus in the research study, school cafeteria lunches were changed to low-fat and low-sodium meals. The success of this change reflected the change in policy, rather than a primary change in consumer behavior. The USDA has recently published recipes recommended for school lunch programs that are lower in total fat, SFA, cholesterol, and sodium than previous recipes. In some parts of the country such as New York State and Massachusetts, programs that include low-fat choices have been developed. Several studies of changes in school nutritional environments, food service, and health education to favor "heart-healthy" eating patterns have been reported.[568]

In the school environment, the behavior of teachers can also be influenced. In a worksite intervention[569] in a Dallas school district, pre- and post-screening and a 10-week behavior-oriented class were offered to teachers. Significant reductions occurred in 11 factors related to cardiovascular disease. Job satisfaction, freedom from worry, energy level, life satisfaction, cheerfulness, relaxation, emotional control, and general well-being all improved.

d. Health Care Facilities

Several national reports[410] recommend that patient contacts with health professionals include dietary education and counseling. National surveys, however, indicate that fewer than 30 percent of men and women report that food habits were discussed when they visited a doctor or health professional.[402,503,570] In a study of medical residents in a university-based training program concerning compliance with eight recommended preventive screening practices, the physicians performed only three of the practices (Pap test, breast examination, and influenza vaccination) as much as 20 percent of the time.[571] A recent report by the Food and Nutrition Board of the National Academy of Sciences indicated that many nutrition education programs in U.S. medical schools are inadequate.[572] On the other hand, there is some evidence that the treatment practices of family practice residents can be improved with education, feedback, and monitoring.[573]

Physicians' Offices. Physicians are often viewed as expert sources on most health topics and as accurate sources of health information.[574,575] Studies of physicians and their role in cholesterol reduction fall generally into three categories: (1) studies of physicians' attitudes and practices regarding cholesterol management, (2) studies of interventions in physicians' offices, and (3) investigations of methods to increase the likelihood and effectiveness of physician management of cholesterol. Ellison[576] has reviewed practice patterns, pointing out problems that require attention.

Studies of the attitudes and activities of family practice physicians show that most physicians report giving some nutrition advice to patients visiting them.[577] However, almost half of these physicians give advice about dietary fat, sodium, and fiber to fewer than 20 percent of their patients. Only 10 percent of physicians give advice to more than 80 percent of their patients. Half of physicians reported not giving dietary advice because the patient did not have an elevated blood cholesterol level. Other reasons included expectations of patient noncompliance and perceived lack of need for dietary fat reduction because there was no weight problem. About one-third reported not being convinced of the value of saturated fat reduction and an equal proportion indicated that a lack of patient interest prevented them from making recommendations. Physician perspectives concerning cholesterol have undergone significant change.[578]

Public expectations may influence physicians behavior. For example, physicians in the Pawtucket Heart Health Program identified requests from the public as an important factor significantly more than physicians in a comparison city.[579] These and other studies indicate that physicians' treatment practices are the outcome of multiple influences.

At the First National Conference on Cholesterol in November 1988, many of the presentations focused on physicians and physician-based interventions.[573,580-582] In a survey of Vermont physicians' attitudes toward screening and dietary advice, Secker-Walker et al.[583] reported that physicians decide which patients will receive dietary advice according to their perception of patient interest. Many believe that nutrition education is the responsibility of the physician and does not require referral to a registered dietitian or nutritionist. These Vermont physicians reported that 35 percent of patients are regularly screened and only 23 percent receive dietary advice. These results are similar to those reported 4 years earlier by Kottke and colleagues.[577] Neither the effectiveness nor the cost of physician nutritional advice has been thoroughly evaluated.

A study of physician-based intervention was conducted in two multiple-specialty clinics (one intervention clinic and one control) in Minnesota to determine the effect of systematic screening and educational intervention for hypercholesterolemia. Patients, whose cholesterol level was in the high- or moderate-risk group according to NIH Consensus Development Conference[275] guidelines, were invited by the intervention clinic to attend a two-session nutrition class. Of those invited, 27.5 percent attended at least one class. In the control clinic, participants were invited to come only for a repeat dietary and cholesterol assessment; a similar recruitment rate was achieved. After 6 months, there was a significant fall in cholesterol (from a mean of 273.4 mg/dL to 248.9 mg/dL; 7.07 mmol/L to 6.44 mmol/L) and a significant change in diet in the intervention group.[584] In a second Minnesota study of cholesterol management in five physicians' office practices, researchers reported that physicians thought that the program was satisfactory, workable, and helpful to patients, and that it improved the clinic's image and reduced cholesterol. All reported that they would continue the program.[585, 586]

In a study of physician office intervention in California, patients within a family practice clinic were recruited, had their blood cholesterol assessed, and were randomly assigned to a treatment group or delayed treatment control group. All patients received standardized instructions from their physicians. Patients in the special treatment group subsequently received computer-based feedback on a dietary assessment, and approximately 70 percent of the patients returned for a 6-month assessment. There were marginally significant changes in blood cholesterol between intervention and control patients, but there were significant improvements on 6 of the 11 targeted food items.[580]

Some insight into the components of a successful office-based cholesterol management program was gained from a study of dietary reduction of hypercholesterolemia by Crouch and colleagues.[587] They reported that multiple face-to-face visits and telephone and mail contacts over 4 months produced comparable and significant reductions in cholesterol, whereas one-time counseling or no contact produced no change or changes in the wrong direction.

Other recent studies have investigated strategies to increase physicians' treatment of high blood cholesterol. Rhode Island researchers, for example, studied three family practice residency programs. In one, residents were trained in blood cholesterol measurement using a fingerstick and desktop

analyzer, a dietary assessment technique, the NCEP guidelines, and followup protocols in dietary and drug treatment. In addition, these residents were given feedback on their performance from a review of charts.[587] The other two programs served as controls. Early results from the intervention group indicate a 100 percent increase in blood cholesterol measurement over 1 year and a threefold increase in referrals for nutrition counseling. In another innovative investigation of methods to improve physicians' cholesterol management practices, hospital house staff were randomized into one of four treatment groups.[588] Treatment groups received either a cholesterol reminder checklist in patient charts, performance feedback of management for each high-risk patient, or both feedback and checklist. Those in the feedback group showed a significant increase in noting elevated cholesterol levels, giving orders for diets to lower blood cholesterol, and ordering lipid profile tests. The combined (checklist and feedback) group showed smaller improvements. In another hospital-based education program, awareness of cholesterol was assessed by notation in charts, the awareness level (20 percent) did not change over 2 years in spite of changes in laboratory criteria for cholesterol. After a 1-hour lecture on cholesterol management, awareness shifted to 69 percent 2 weeks after the program and to 52 percent 6 weeks later.[589]

In San Francisco, researchers achieved a 35 percent recruitment rate of community physicians to participate in a study of office-based cholesterol management practices. Reasons for participation included personal interest, patient demand, availability of continuing medical education credit, and willingness to contribute to research. Those who refused (65 percent) gave lack of time, interest, and financial incentive as reasons for nonparticipation.[590]

These studies suggest that physician interventions can be effective in helping people change their eating patterns and, at times, lower their blood cholesterol level. Concern about the cost-effectiveness of such efforts has been expressed by Weinstein and Stason[591] in a review of five studies, and subsequently by Disbrow.[506]

Other Health Professionals and Other Health Care Settings. In recent years, group practices, health maintenance organizations, and other forms of practice have brought large groups of patients into prevention-oriented health care settings where they are seen by teams of providers. These changes, along with changes in reimbursement methods, have led to consideration of more cost-effective methods for delivering health care.

The role of other health professionals such as nurses, registered dietitians, pharmacists, and social workers has been considered in providing lower-cost nutrition and health education. Three recent papers examined cost-effectiveness of blood cholesterol lowering using various mixes of health professionals.[506,592,593] The analyses of various factors, such as salary, fringe benefits, overhead costs, and numbers of clients, clearly show that nonphysician health professionals can provide individual and group nutrition counseling at relatively low cost.

Many of the studies reported here were pilot projects and lack long-term followup. However, several tentative inferences can be drawn. First, blood cholesterol management in physicians' offices and other health care settings is accepted and approved by patients. Second, physicians and other health professionals will participate in and maintain cholesterol management programs, even though they may need additional stimuli to initiate such programs on their own. Third, the numbers of physicians and other health professionals practicing vigorous cholesterol management can be increased. Health professionals are likely to respond positively to patient interest and demand for advice about high blood cholesterol management and diet. Finally, the entire health community should join in the effort to find cost-effective ways to help people adopt new eating patterns and decrease blood cholesterol levels.

Health Facility Food Services. Many hospitals and other health facilities provide food to large numbers of people. Despite the opportunity to model food choice behavior to achieve low blood cholesterol levels, little research has been reported in this area other than the National Institutes of Health study previously described.[485,486]

5. Community-Level Interventions

This section reviews three kinds of interventions: media-, policy-, and community-based.

a. Media-Based Interventions

The media influence community practices regarding politics, economics, and public health. Media attention to disease prevention and health promotion can influence the status of health as a topic of community importance.[478] The media are particularly powerful at influencing individual behavior directly through mass media campaigns and indirectly by influencing public opinion, prioritizing issues, and helping to create and reinforce social standards.

Mass Media Campaigns. Mass media campaigns are one important means of influencing the information environment, and they in turn supplement the effects of other social, organizational, network, or individual change efforts.[594-596] One among many examples of media campaigns designed to change a particular dietary habit is the National Cancer Institute's (NCI) Cancer Prevention Awareness Program. This nationwide program to prevent cancer seeks to change knowledge, attitudes, and behavior through information and education for the general public and health professionals. A 2-year cereal advertising campaign, which included NCI's diet message and telephone number in television ads and on the backs of boxes of a bran cereal, produced more than 20,000 telephone and 30,000 written inquiries to the NCI. A study of cereal sales in the greater Washington, D.C. area revealed that the sales of all high-fiber cereals increased, not just the brand advertised.[596-598]

b. Policy-Based Interventions

The laws, policies, and allocation of public resources at all levels of government can have important impacts on the health of the public. Alcohol and cigarette taxes and seat belt laws are but a few examples of the potent effect of government regulation. Local or national policy changes can also influence eating patterns.[599] The effects of wartime food rationing serve as one example. Another is the recent change in the term for grading lean beef from "good" to "select," a policy change that may influence consumers to choose leaner meat.[11,600] In terms of food choices, the food industry, wholesale and retail food vendors and food service establishments, and public health agencies are influences that are increasing the availability of foods that are low in calories, fat, SFA, cholesterol, and sodium. These groups can further support activities such as education of food servers, grading, and labeling of foods.[503] The efforts of the National Livestock and Meat Board to stress availability and consumption of smaller, leaner meat portions represent a helpful organizational policy change.

Regulation affects the actions of organizations. Using regulations as an incentive for health promotion, a community in Pennsylvania allowed food service institutions to receive reduced fees if they adopted healthful policies in their food outlets. These policies included designated nonsmoking areas, certification of a percentage of employees in cardiopulmonary resuscitation, and attaining certification in food sanitation principles and practices.[601] While not all of these policies were food related, this study exemplifies the ways that communities can stimulate and support organizational adoption of healthful policies.

c. Community-Based Interventions

A growing number of community-based studies have been reported or are underway. The Stanford Three Community Study (TCS) and the Finnish North Karelia Project[343,344,346,527,602-605] achieved similar cholesterol, blood pressure, and smoking results after 3 years of education in the TCS and after the first 5 years of education in the North Karelia Project. In the Finnish study, less cardiovascular disease morbidity and mortality were reported after 10 years in comparison not only with the adjoining county but with the remaining part of Finland. Preliminary results from the Stanford Five City Study suggest favorable trends.[351] Results from two other large Heart Health Studies in

Minnesota and Rhode Island, discussed previously, will be available in the early 1990's. Four other community-based studies using methods similar to the TCS and the North Karelia Project have reported their main findings. Only one of them, in three small rural South African towns, reported significant decreases in blood cholesterol and blood pressures as well as in smoking.[606] Two others, a four-town study in Switzerland and a three-town study in Australia, reported significant drops in smoking rates.[607,608] The favorable effects of these studies present an encouraging picture for community-based intervention.

6. Conclusions

The foregoing section has reviewed investigations of interventions targeted at individuals, networks, organizations, and communities/societies. Studied strategies for change have included face-to-face persuasion, skills training, information dissemination, minimal contact programs, use of incentives, policy and regulatory change, and use of influential individuals. The channels for communicating dietary information have included use of the media, new technologies, printed materials, health professionals, and other credible sources.

Some intervention strategies have been more rigorously evaluated than others, some have more successfully integrated behavior theory than others, and some have used more formative research in the development and implementation of interventions than others. Not all of the potential points of influence have yet been investigated in the effort to improve the diets of Americans. Nonetheless, many feasible and well-evaluated interventions do provide scientific support for recommendations designed to influence national eating patterns.

7. Summary

The data reviewed here and elsewhere[5,11] implicate elevated blood cholesterol in the pathogenesis of atherosclerosis, especially CHD. Furthermore, while there is evidence that blood cholesterol levels are affected by many factors, food habits are among the most important. The strongest dietary relationship with blood cholesterol level is between saturated fatty acid intake (especially those SFA containing 12 to 16 carbon atoms) and elevation of both total and LDL-cholesterol levels. The average current U.S. consumption of saturated fatty acids (14 percent of total calories) is high and warrants a significant reduction. A recommendation that less than 10 percent of calories be derived from SFA is based on epidemiologic evidence that healthy populations with SFA levels of 10 percent or less have low CHD rates and on a large body of evidence from many kinds of studies that reducing SFA will lower the level of blood cholesterol and thus lower coronary risk.

PUFA and MUFA also influence circulating lipids. Their major effect derives from their substitution in the diet for SFA. The data on which to base quantitative dietary recommendations concerning PUFA or MUFA are less clear than are those for SFA.

Given current knowledge, the best approach appears to be to emphasize a reduction of SFA, as above, and of total fat. Several beneficial effects can be expected. Reducing total fat facilitates attainment of the SFA goal. Limiting all fat intake may also help to prevent obesity, an important factor in control of high blood cholesterol, and some forms of cancer.[5] Finally, presenting quantitative recommendations for SFA and total fat is likely to enhance public comprehension and compliance.

High levels of dietary cholesterol have, in most people, some impact on blood cholesterol level. The evidence for limiting intake is strong. A reduction to an average cholesterol intake below 300 mg per day will produce a significant reduction in the average blood cholesterol and, correspondingly, an important anticipated decrease in CHD rates. While achieving even lower cholesterol intake would likely lower CHD even more, a target of 300 mg per day appears to be realistic in light of the current eating pattern of many people.

VI. Ethnic, Cultural, and Minority Characteristics That Influence Diet and Health

A. Introduction

It is a truism that people eat food, not nutrients. The food we consume determines our energy and nutrient intakes. The adequacy of our diets is a consequence of the food we select and its nutrient content.

Culture, society's design for living, is a major determinant of food habits in any society. Cultural values help determine what edible items are selected from the environment, and how they are transformed into what the society considers food. Customs govern the preparation, flavoring, presentation, combination, order, and eating rituals relating to food. Violation of these customs by well-meaning health professionals or others seeking to reform food habits may threaten community identity and solidarity, and may result in rejection of the advice.

Therefore, to be effective in the long run, public health efforts need to encourage changes in those customs and values that promote excess consumption of fat, saturated fat, and cholesterol.(409) And in the short term, specific guidance, to be acceptable, must respect cultural customs and work within them. Public health approaches must also consider risk of malnutrition versus risk of CHD in groups where poverty and social isolation are factors.

The following section deals with the many factors that influence diet and health in all populations, especially in ethnically diverse or minority groups. Caution is indicated when interpreting health data because ethnic and cultural factors are highly confounded with economic and educational status and with access to both preventive and curative health care.

1. Dynamics of Culture Change

In order to understand the characteristics of ethnic, cultural, and minority groups that may influence intervention efforts, one needs to examine the dynamics of culture change. American eating habits and traditions reflect the many customs brought to the country by immigrant groups.

During the immediate post World War II period, as a result of a new national consciousness, technological developments in mass communication, and a rapidly expanding food industry, a "national diet" emerged. It synthesized many elements into the lowest common denominator, or what has been otherwise described as the "blanderizing of America." Reaction to this "leveling" process began in the late 1960's. The food industry reaction has been one of market segmentation, and the growth of conveniently packaged ethnic specialties and of ethnic restaurants. These developments have created an environment of greater flexibility and have weakened some of the cultural rules governing food selection and preparation. This flexibility and greater respect for ethnic foods by the majority culture provide an opportunity to enhance intervention for dietary change.

2. Health Belief Systems

An important component of culture is how the society organizes and processes information. Understanding this process is crucial to communication. The belief system of the majority American culture is strongly rooted in the scientific method. This is reflected in the Western system for treatment and prevention of illness. It is also evident in delineation of food groups based on their nutrient content. A common and persistent alternative health belief system, among such diverse populations as some in Latin America and Asia, is the hot-cold theory or yin-yang. According to this theory, health is maintained by the proper balance of hot and cold factors. Foods are classified as hot, cold, or neutral. A dietary intervention strategy developed within this framework might be far more effective than nutrient-based food groups in sub-populations that adhere to the hot-cold theory. It is not necessary to adopt Western thinking in all respects to preserve or promote good health. In fact, many cultural rules for food use in traditional societies are directed at perceived health benefits.

Other cultural characteristics may also affect intervention strategy. Weaver[6-10] has proposed a model for comparing two basic culture-and-personality systems. It represents two ways of organizing society, its values, harmony, and perceptions. In Weaver's model, cultures are placed along a continuum ranging from abstractive to associative worlds. Mainstream American culture falls on the side of the abstractive world. Ethnic minorities may fall anywhere along the continuum depending on the degree of acculturation. Since many of the factors affect communication, it is important to recognize differences that may exist among ethnic minorities. For example, according to Weaver, the abstractive world is loosely integrated, urban, and heterogeneous; social structure is based on achieved status, and the nuclear family is important; there is a high degree of social and physical mobility. Basic values include change/action and individualism. Interaction is written, electronic, and impersonal, and time is future or outcome oriented. By contrast, the associative world is based on a highly integrated homogeneous community whose roots are rural. The social structure is based on ascribed status, kinship, and the extended family. There is little social mobility. Basic values include stability, harmony, and the sense of belonging. Interaction is face to face, personal, verbal, and nonverbal. Time orientation is past-present. The model provides a theoretical framework for understanding cultures, which is crucial to designing effective strategies for ethnic minority groups in the United States.

Intervention strategies in groups with more associative characteristics should use kinship and community resources as much as possible. Planners must take into account the implications of past-orientation for an intervention whose outcome is future oriented. At an extreme, looking into the future may be seen as prophecy and thus to violate religious principles. However, among the tenets of most major religious and ethical systems is a moral responsibility to maintain a healthy body.

3. Acculturation

Acculturation is the process whereby the characteristics (design for living) of the dominant culture are adopted and assimilated. The process is complex, varies among individuals, and depends on many factors, including the extent and duration of exposure to the dominant culture. Low levels of educational attainment, lack of proficiency with the language of the dominant culture, social isolation, subtle discrimination, low income, and high unemployment retard acculturation.

4. Poverty

Many ethnic minorities have a substantial proportion of educationally and economically disadvantaged persons (table 6). For some groups, this may be a temporary situation, whereas for others it may become a long-term reality. Much has been written about the so-called culture of poverty. Adherents to this theory contend that the culture of poverty has certain characteristic traits including strong feelings of fatalism, helplessness, dependence, and inferiority. The poor, it is claimed, have a strong present-time orientation and thus do not defer gratification or plan for the future. They often believe in male superiority and are sensitive to external status symbols. Once the culture of poverty comes into existence, some suggest that it often persists in succeeding generations.[6-11] In any

Table 6

Sociodemographic Characteristics of Population Subgroups in the United States

Characteristics	**Ethnic/Racial Background**				
	White	**Black**	**Hispanic**	**Asian/Pacific Islander**	**Native American**
Total Percent Distribution (1980)	79.6	11.5	6.4	1.6	0.9
Median Age (1980)	31.6	24.9	23.2	28.7	22.4
Percent High School Graduates (1980)	88	79	58	75	31
Median Household Income	$23,270[†]	$13,270[†]	$16,228[†]	$22,713[*]	$15,900[*]
Percent Below Poverty Level	11[†]	34[†]	30 ‡	13.1[*]	29[*]
Percent Unemployed	8.6 ‡	18.9 ‡	13.8 ‡	4.7[*]	13 ‡
Average Fertility Rate (1980)	1.7	2.3	2.3	--	4.0
Percent of Households Headed by Women (1980)	10.9	37.7	23	11	24

[*] 1979

[†] 1981

‡1982

Source: Adapted from the *Report of the Secretary's Task Force on Black and Minority Health,* Volume I.[307]

event, sensitivity to the psychosocial needs of the poor and the dignity of the individual is important in intervention programs. In this country, more women than men are poor.

B. Characteristics of Important Population Subgroups

Undoubtedly the summaries of the important population subgroups that follow reflect the influences of a mix of the original cultures, the culture change process, health belief systems, and socioeconomic and educational status.

1. Blacks

a. Social and Demographic Characteristics

Blacks, the largest single minority, represent nearly 12 percent of the U.S. population (table 6, page 95). The relatively low median age of 24.9 years reflects both birth rates and slightly shorter average life expectancy. Blacks have the highest rate among minorities of high school completion, but lag behind Anglos in receipt of high school diplomas. The median income is lower for blacks than for any other group, with 34 percent living beneath the poverty level. Further, the average unemployment rate is substantially higher than that of other major minority groups. Household size is larger among blacks than among Anglos, and nearly 40 percent of black households are headed by a woman.

b. Health Indicators

According to the data summarized from 1979-81,[307] 59,000 excess deaths occur each year among blacks compared with mortality rates for whites. Homicides alone account for 38 percent of the excess deaths among black males younger than 45 years of age. Cardiovascular disease and cirrhosis account for another 18 percent, and infant mortality for 23 percent. The infant mortality rate among blacks is twice that of whites. For black females younger than 45 years of age, infant mortality is responsible for 35 percent of the excess deaths, cardiovascular disease accounts for 17 percent, and homicide for 14 percent. Among black males younger than 70 years of age, the three leading causes for excess deaths are cardiovascular disease (24 percent), homicide (19 percent), and cancer (16 percent). For black women younger than 70 years of age, cardiovascular disease accounts for 41 percent of excess deaths and cancer for 10 percent.

In 1986, age-adjusted mortality rates from CHD were about 8 percent lower for black males than for white males. Age-adjusted CHD mortality rates for black women were almost 20 percent higher than for white women.[612]

c. Risk Factors for Coronary Heart Disease in Blacks

A National Center for Health Statistics report revealed that the mean total blood cholesterol levels in black and white adult men and women are similar,[613] whereas the HDL-cholesterol levels are higher in black men than in white men.[307] Cigarette smoking prevalence is greater among blacks than whites, but the prevalence of heavy smoking is greater among white than black adults.[614,615] Obesity is also common in black women and may provide a partial explanation for their excess coronary disease risk.[616,617] Blacks have a higher prevalence than whites of high blood pressure and of diabetes. Each of these risk factors causes roughly similar increases in risk for blacks and whites.

d. Eating Patterns Among Blacks

According to the 1985 Continuing Survey of Food Intakes by Individuals (CSFII), dietary intakes of fat, saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids, expressed as average percentages of total energy, are similar for black and white women. Data from several surveys show that black adults on average consume fewer calories and more cholesterol than whites.[14-16,618] Mean intakes of some essential nutrients for black women, expressed as a percent of the RDA, are lower than for white women. As shown in the CSFII, mean intakes of vitamin B_6, folacin, calcium, magnesium, iron, and zinc are lower for black than for white women, but only for calcium and magnesium are the differences such that black women are likely to be at much greater risk of deficiency compared with white women.[14-17] Intakes of these nutrients by black men are lower than by white men, but black men achieve greater than 70 percent RDA average levels for all nutrients except folacin.[17] Although the CSFII sample of blacks contained a larger proportion of individuals below the poverty level, living in central cities, and residing in the Northeast and South, the differences in intakes of essential nutrients do not appear to be attributable to the sample selection.

Mainstays of traditional black food patterns have tended to reflect a southern rural heritage, i.e., fried, barbecued, roasted, or smoked pork and chicken; rice and grits; dried legumes; sweet potatoes; hot breads (biscuits, corn bread, and rolls); buttermilk; custards; and vegetables seasoned with smoked bacon, salt pork, ham hocks, or neck bones. In one study, use of some foods apparently was related to perceptions of social status. Recipients of public welfare considered some "modern" items, such as unseasoned vegetables and variety in meals, beyond their reach.[619] Traditional black recipes are sometimes modified to suit modern tastes[620] as part of a process of acculturation, together with avoidance of many fatty, fried, and overcooked foods. It is likely that the food patterns of blacks vary according to the economic, regional, and social influences of communities. Thus generalizations are inappropriate.

2. Hispanics

a. Social and Demographic Characteristics

Hispanics are the second largest minority in the United States, with approximately 18.9 million residents in 1987 or 7.5 percent of the United States population.[307,621] The Hispanic growth rate is now the fastest of any population group in the United States. The Hispanic minority has four major subgroups: Mexican-Americans at 10.9 million (58 percent); Puerto Ricans at 2.7 million (14 percent); Cubans at more than 1 million (6 percent); and other Latinos at 4 million (22 percent) including Dominicans, Haitians, and others from South and Central America. Of this Hispanic minority, 60 percent live in California, Texas, Colorado, New Mexico, Arizona, and Florida, and 50 percent live in cities of more than 1 million in population. The Hispanic population has a median age of 23 years.

As of 1980, only 58 percent of Hispanics had earned high school diplomas, and 10 percent had graduated from college. In 1981, the median income for Hispanic families was $16,228. Thirty percent of Hispanics lived below the poverty level in 1982, and nearly 14 percent were unemployed.

Hispanics have relatively large families, averaging 2.3 children per family. Fifty percent of all Hispanic families have five or more children. Twenty-three percent of all Hispanic households are headed by women; among Puerto Rican households the proportion is even higher, at 40 percent.

b. Health Indicators

National mortality rates among Hispanics are unknown in the absence of a national data collection system. Many states with substantial numbers of Hispanic residents (e.g., Florida and New Jersey) have no Hispanic identifier on death certificates. However, Spanish surname data from Texas and national death certificate data listing Mexico or Cuba as the country of birth provide the following information. Among Mexican-born Hispanic males, homicides and unintentional injuries account for 98 percent of the total excess deaths. Cirrhosis accounts for the other 2 percent. For Mexican-born women, homicide and diabetes accounted for 100 percent of the total excess deaths.[307] Hispanic men and women appear to have CHD mortality rates that are 5-15 percent lower than those of non-Hispanics.

c. Risk Factors for Coronary Heart Disease in Hispanics

Diabetes mellitus is a major health problem for Hispanics, especially for Mexican-Americans and Puerto Ricans.[622,623] The prevalence of obesity, noninsulin-dependent diabetes, high LDL-cholesterol and low HDL-cholesterol levels in Hispanics might be expected to increase their CHD risk. The prevalence of high blood pressure and physical inactivity is higher in Hispanics than in non-Hispanics. However, the scarce data that exist indicate that Hispanic males have rates similar to those of other white males. Because the Hispanic categorization includes many different populations, available data on the prevalence of elevated blood cholesterol are difficult to interpret. Cholesterol levels have been reported as being higher in Hispanics than in non-Hispanic whites in Texas, equal to those of non-Hispanic whites in California, and lower than those of non-Hispanic whites in Puerto Rico.

Mexican-Americans with low socioeconomic status (SES) and low levels of acculturation have significantly worse CHD risk factor profiles than those in higher SES groups.[624,625]

d. Eating Patterns Among Hispanics

Hispanics are not a homogeneous group, either in heritage or food patterns. Dominicans, Mexican-Americans, Cuban-Americans, and Puerto Ricans have distinctive native cuisines. The impact of these differences on nutrient intake is not well documented. Many traditional dishes are complicated and time consuming to prepare and hence tend to be reserved for special occasions. Traditional core dietary constituents such as rice, beans, cornmeal, squash, and other starchy vegetables and chicken are compatible with cholesterol-lowering dietary recommendations.

3. Asian/Pacific Islanders

a. Social and Demographic Characteristics

Asian/Pacific Islanders accounted for an estimated 1.6 percent (3.7 million) of the total population in the United States in 1980,[307] although the actual population has increased substantially because of the immigration of Indochinese under the Refugee Resettlement Program. In 1980, 58 percent of Asian/Pacific Islanders were foreign born, the largest proportion of all minority groups. Asian/Pacific Islanders are also the most diverse minority group, originating from more than 20 different countries. The three largest subgroups are Chinese (812,000), Filipinos (781,000), and Japanese (716,000). The data on Asian/Pacific Islanders are heavily influenced by these three largest subgroups, which would mask important socioeconomic characteristics of the more recent Indochinese immigrants.

According to the 1980 census, 56 percent of Asian/Pacific Islanders lived in the western United States. Thirty-three percent lived in California and 10 percent lived in Texas. The median age of Asian/Pacific Islanders is higher than that of other minorities, at 28.7 years.

The median education level of Asian/Pacific Islanders is similar to that of nonminorities. Of Asian/Pacific Islanders, 75 percent have completed high school, and approximately 33 percent hold a college degree.

In 1980, the median household income for Asian/Pacific Islander families was higher than that of any other U.S. minority, at $22,713 (see table 6, page 95). However, this figure masks the true extent of poverty as it often represents the total of several incomes when families share houses. Median income levels vary substantially between groups, with the lowest, $12,840, being earned by the Vietnamese. The unemployment rate among Asian/Pacific Islanders in 1979 was 4.7 percent, and the poverty rate was 13.1 percent. Only 11 percent of Asian/Pacific Islander families are headed by women.

b. Health Indicators

Compared with all other population groups in the United States, Asian/Pacific Islanders are on average reported to be the healthiest and have the lowest risk of early death.[307] For the entire population, there are no excess deaths reported for any cause. However, substantial variation does exist regarding the relative health status among Asian/Pacific Islander subgroups. For example, native Hawaiians have excess deaths owing to cardiovascular disease, cancer, diabetes, infant mortality, and accidents. Other Asian/Pacific Islander groups such as recent immigrants from Southeast Asia have higher rates of tuberculosis, hepatitis, and anemia.

c. Risk Factors for Coronary Heart Disease in Asian/Pacific Islanders

In their native countries, Asians tend to have low blood cholesterol levels, a low prevalence of obesity, and a low prevalence of CHD. As immigrants move from areas of low to high CHD prevalence, they exhibit risk factor levels that approach those of the host-country population. For example, a comparison of risk factors in Japanese men with risk factors in Japanese-American men living in Hawaii and California showed clear gradients in blood cholesterol—181 mg/dL (4.68 mmol/L) for the Japanese, 218 mg/dL (5.64 mmol/L) for their counterparts in Hawaii, and 228 mg/dL (5.90 mmol/L) for the Japanese-Americans in California.[342] Moreover, the Japanese men had lower intakes of SFA (16 g/day) and dietary cholesterol (464 mg/day), compared with their counterparts in Hawaii (SFA, 59 g/day; cholesterol, 545 mg/day) and California (SFA, 66 g/day; cholesterol, 533 mg/day). Similar changes could be expected for other Asian immigrants to the United States.

d. Eating Patterns Among Asian/Pacific Islanders

Asian Americans are a heterogeneous group; however, the nutritional habits of Asian/Pacific Islanders can be generalized to some extent. Rice is a primary source of calories, although the varieties used are often culturally specific.[307] On the average, Asian/Pacific Islanders, compared with other groups, consume more vegetables, fruits, fish, and shellfish and consume less meat and dairy products. Different flavoring ingredients, cooking methods, and cultural rules governing the manner of service make each group's diet distinctive. As with many other ethnic groups, many traditional foods are receiving less emphasis and are being replaced by popular American foods. Hence, secondary and tertiary generations of Asian/Pacific Islander immigrants are becoming increasingly acculturated to nonminority dietary practices and, as a result, the consumption of animal proteins, fats, and refined sugar is on the increase.

4. Native Americans

a. Social and Demographic Characteristics

The native American minority is heterogeneous. Although it is composed of American Indians, Aleuts, Alaskan Eskimos, and native Hawaiians, the data reported from most national surveys refer primarily to American Indians and Alaskan natives.[307] According to the 1980 census, the native American minority is the smallest of all four groups—its total population of approximately 1.5 million represents less than 1 percent of the total U.S. population.[307] Fifty percent of native Americans live in the West or Southwest. Twenty-four percent live on reservations, and 8 percent live on historic trust sites. Most reservations have fewer than 1,000 inhabitants, and only one reservation has a population exceeding 100,000. Tribal customs vary considerably.

The median education level among native Americans is lower than that of all other minority groups. Only 31 percent of native Americans have graduated from high school, and only 7 percent hold a college degree. The median family income among native Americans in 1979 was $15,900. Twenty-nine percent of native Americans lived below the poverty level in 1979, and, in 1982, 13 percent were unemployed.

The average native American family has 4.0 members, the largest of any population subgroup in the United States. In addition, native Americans have the highest total birth rate—approximately twice that of the nonminority rate. Twenty-four percent of native American households are headed by women.

b. Health Indicators

The native American life expectancy is 6 years less than that of nonminorities, and the median age is the lowest of all groups, at 22.4 years.

Native American males under the age of 70 years experienced a 28 percent excess in deaths in 1979-81.[307] The three primary causes of the excess deaths were accidents (44 percent), cirrhosis (13 percent), and homicide (8 percent). Native American women younger than 70 years of age also experienced a 28 percent excess death rate. The three major causes of excess mortality were accidents (29 percent), cirrhosis (22 percent), and infant mortality (9 percent). Heart disease is a significant contributor to all-cause mortality of native Americans but is proportionately less a contributor in this subgroup than in the general population. The health of native Americans is a serious problem that must be addressed with a great deal of skill since these individuals have a special relationship with the Federal Government.

c. Risk Factors for Coronary Heart Disease in Native Americans

Both obesity and diabetes are major public health concerns for native Americans,[307] especially among Southwestern desert tribes. Prevalence estimates of obesity and diabetes as high as 75 percent have been reported. Alcohol use is widely prevalent. The data indicate a lower prevalence of elevated blood cholesterol, high blood pressure, and physical inactivity in native Americans than in the majority population.

d. Eating Patterns Among Native Americans

Native Americans are a culturally diverse group. Data on the nutrient intakes of native Americans are scarce. A common impression is that the diet is high in fat. However, in one study of Navajo women, fat contributed only 30 percent of calories, mostly saturated (animal) fat.[626] Another study in women indicated a fat level of about 37 percent of calories.[627]

Dietary patterns are highly variable, depending on the tribe, geographic location, and economic resources. Although most traditional foods are preferred and continue to be a source of pride, they are reserved mostly for ceremonial occasions.[611,620,628] Many elements of native American diets such as corn, beans, squash, wild game, fish, and wild plants are health promoting. There are Federal programs on reservations (e.g., EFNEP) that are targeted toward helping native Americans follow the dietary guidelines using traditional foods and commodities supplied by the Federal Government.

5. Summary

More systematic investigation and documentation of the health, nutritional status, food practices, and nutrient content of native foods of minority groups are needed. In addition to food and nutrient intakes of individuals, information on the importance of particular foods, preparation techniques, format of meals, health beliefs, and related social and ecological factors should be collected, to help in designing strategies relevant to cultural practices.[629]

VII. Screening the Population for Blood Cholesterol Level

The development of consensus and recommendations by the scientific community for the definition and management of high blood cholesterol plus the advent of new technologies for blood cholesterol measurement have resulted in numerous blood cholesterol screening programs both within health care facilities and in the community. Many in the population have been screened outside health care settings, and many more will be in the coming years.

Public cholesterol screening that meets NCEP standards[22] has value as a supplement to the measurement of blood cholesterol that occurs as part of routine health care. Adherence to these standards will minimize the risk that poor screening programs will be conducted that fail to measure cholesterol accurately or educate screenees adequately about cholesterol, or that lead to inappropriate referral and/or false reassurance.

The rationale for public screening for blood cholesterol is found in two major areas. The first of these relates to the educational value of these programs. Public screening programs must provide screened individuals with health information about cholesterol beyond the simple knowledge of their "cholesterol number." This information is available in the form of printed documents. The presence of knowledgeable staff to answer reasonable questions from participants is also important. Documents should include a description of the meaning of a single blood cholesterol measurement, the importance of a prudent diet in lowering blood cholesterol, specifics about such a healthy diet, and the appropriate followup steps that individuals should take depending on their blood cholesterol level and other CHD risk factors.

The second major role that public screening programs can play is the traditional case-finding function. Public screening programs can be particularly helpful in detecting individuals with elevated blood cholesterol levels who might not be detected by the health care system. This includes young adults, middle-age males, individuals with low income or education, and minorities. Public screening programs should target their efforts to populations that have high proportions of these underserved individuals.

One of the dangers of public screening programs is the tendency of some individuals to use them as an alternative source of medical care. This is inappropriate. Screening programs should not be a substitute for a health care visit, nor should they replace routine medical monitoring of blood cholesterol levels for anyone already under treatment. While self responsibility for control of cholesterol is important, issues such as drug side effects, changes in therapy, and complications require the judgment and experience of a health care professional. Public screening programs, except in rare instances, are not equipped to provide this medical advice.

Public screening programs should provide blood cholesterol readings at reasonable cost. It is recognized that any recommendation that targeted screening should include low-income and low-education groups means that subsidization by public and private resources may be necessary to encourage these individuals to attend a screening program. In addition, efforts must be made to develop sources of referral and followup that provide care for these target groups.

Only the measurement of total blood cholesterol is currently recommended for public screening programs. New technologies for the direct measurement of LDL- and HDL-cholesterol and the potential benefit of such measurements have been considered by the panel. At present, reliable measures for field settings, while under development, are not yet available. Because of concerns about cost, the meaning of a single HDL-cholesterol measurement, and quality control, the panel does not recommend HDL-cholesterol measurement as presently suitable for public screening efforts.[196] When better technology becomes available and has validated accuracy and precision, it may become appropriate to add this measurement.

Estimation of the effects of screening and treatment for cholesterol on the costs of health care is important. Reasonable projections may rely on methods similar to those used for hypertension[591] and could be based on the results of epidemiologic research and on the various trials that have examined the efficacy of cholesterol-lowering agents. Cholesterol screening programs, even if they lead to net increases in the costs of health care, are reasonably cost-effective if they are efficiently run, targeted at groups likely to contain high-risk individuals, and successful in achieving long-term reductions in blood cholesterol.

Of particular importance is the need to maintain high quality programs while maximizing the cost-effectiveness of screening programs. Cost-effective cholesterol screening programs will have the following characteristics:

- Program marketing strategies that reach the hard-to-reach;
- Staffing matched to level of program activity;
- Well-organized screening and educational processes;
- Well-trained staff;
- Staff incentives for productivity;
- Low program overhead;
- Equipment whose cost is suitable to needs;
- Screening targeted at high-risk, underserved populations;
- Rescreening limited to pre-established intervals, with avoidance of rescreening "worried well" on demand;
- Accurate cholesterol measurement, to minimize false-positive and false-negative values;
- Clear behavior change messages tailored to the educational level and cultural patterns of the population;
- Referral to medical care limited to high-risk individuals; and
- Followup to ensure successful completion of referral.

Cholesterol screening programs, as well as physicians who treat identified high-risk individuals, are urged to give high priority to opportunities to reduce costs, as well as enhance the effectiveness of their activities.

In October 1988, the NHLBI convened a Workshop on Public Screening for High Blood Cholesterol, to review the available scientific knowledge concerning screening and to develop guidelines for screening. The report from that workshop contains further details.[22]

VIII. Glossary

AHA	American Heart Association
ATP	Adult Treatment Panel
CHD	coronary heart disease
DHHS	United States Department of Health and Human Services
FDA	Food and Drug Administration
HDL	high density lipoprotein
LDL	low density lipoprotein
LSP	Laboratory Standardization Panel
MRFIT	Multiple Risk Factor Intervention Trial
MUFA	monounsaturated fatty acids
NCEP	National Cholesterol Education Program
NCHS	National Center for Health Statistics
NHANES	National Health and Nutrition Examination Survey
NHLBI	National Heart, Lung, and Blood Institute
NIH	National Institutes of Health
NRC	National Research Council
PUFA	polyunsaturated fatty acids
RDA	recommended dietary allowance
SFA	saturated fatty acids
USDA	United States Department of Agriculture

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